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CLINICAL STUDY

Bilateral thoracoscopic cardiac sympathetic denervation in patients with refractory ventricular tachyarrhythmias non-responsive to other treatment

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ABSTRACT

OBJECTIVES: To explore the efficacy and safety of bilateral thoracoscopic cardiac sympathetic denervation (BTCSD) as an underutilised last-resort surgical technique for patients with ventricular tachyarrhythmias and electrical storm non-responsive to other treatment.

BACKGROUND: Patients with refractory ventricular tachycardia, ventricular fibrillation, and electrical storm are at high risk of sudden cardiac death. In some patients, suboptimal results are achieved despite treatment with anti-arrhythmic drugs, implantable cardioverter-defibrillator and cardiac catheter ablation. Minimally invasive surgery affecting the stellate ganglions and sympathetic chain is an additional alternative treatment modality that may help avoid heart transplantation.

METHODS: We present our experience of 3 patients who were treated with this technique for the first time in Slovakia in cooperation with the National Institute for Cardiovascular Diseases. Publications on this issue are scarce despite its potential for specific patients. Modifications to avoid complications derived from our experience of sympathectomies for hyperhidrosis are introduced, and improvements are proposed to promote this technique.

RESULTS: All patients showed a reduction or cessation of arrhythmias and ICD shocks with no periprocedural complications.

CONCLUSION: Our experience showed that BTCSD is a safe and feasible technique with a low complication rate and promising results. The limitation of this paper is the low number of patients in our group *(Tab. 1, Fig. 3, Ref. 25)*. Text in PDF *www.elis.sk*

KEY WORDS: cardiac sympathetic denervation, VATS sympathectomy, ventricular arrhythmia, stellate ganglion, sympathetic nervous system.

Introduction

The autonomic nervous system (ANS) has an essential role in the pathophysiology of ventricular arrhythmias via the cardio-neuronal axis (1). The treatment of ventricular arrhythmias (VA) aims to prevent degeneration of cardiac myocytes, development of heart failure and sudden cardiac death. There are several well-known treatment modalities targeting the ANS with suboptimal results. One of the underused but promising techniques is cardiac sympathetic denervation (CSD), performed by means of video-assisted thoracoscopic surgery. The standard treatment regime includes combined medical therapy with beta-blockers and calcium channel blockers, implanted cardioverter defibrillation (ICD), and cardiac catheter ablation. Other direct but temporary modalities in the acute setting that affect the cardio-neuronal axis are intubation and sedation, thoracic epidural anaesthesia and percutaneous stellate ganglion block that can serve as a bridging technique until more definitive treatment is arranged (2). Patients who are unresponsive or not compliant with this treatment regime and patients with electrical storms are candidates for thoracoscopic cardiac sympathetic denervation (3, 4). Since these patients have a higher risk of sudden cardiac death, CSD provides an effective and safe *dernier ressort* method (5, 6). At our Department of Thoracic Surgery in Bratislava, we perform this technique after the indication is made in cooperation with the Department of Arrhythmias and Pacing of the National Institute for Cardiovascular Diseases in Slovakia. In this paper, we present our early experience of this

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method that was first applied in Slovakia at our department, and for various reasons, it is still underutilised globally.

Anatomy

The sympathetic nervous system contributes to both physiology of cardiomyocyte excitation and pathophysiology of ventricular arrhythmias. The increased sympathetic tone is associated with arrhythmogenesis, while increased parasympathetic tone is believed to be protective. CSD reduces beta-adrenergic automation while prolonging repolarisation and refractory period without affecting the contractile function (7, 8).

Efferent sympathetic preganglionic neurons that control the cardiac function are localised in the intermediate zone of the thoracic spinal cord at the level of segments T1–T4. Their preganglionic axons exit the spinal cord through the ventral horns via the ventral root of the spinal nerve and enter the paravertebral ganglions of the sympathetic chain through the white *rami communicantes* at their respective levels. Within the sympathetic chain, these preganglionic axons synapse on neurons within the stellate ganglion (fusion of inferior cervical and T1 ganglia) and ganglia at spinal levels T2–T4. Postganglionic nerve fibres originate mainly from the left and right stellate ganglia (LSG and RSG), exit the ganglia via grey *rami communicantes* and continue as the cardiopulmonary nerves forming the cardiac plexus at the base of the heart. The stellate ganglion is located at the level of the C7 cervical vertebra, anterior to its transverse process and neck of the first rib (9).

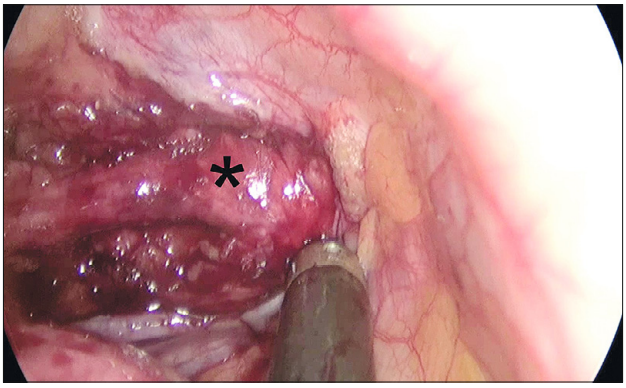

Fig. 2. Dissected stellate ganglion (black star).

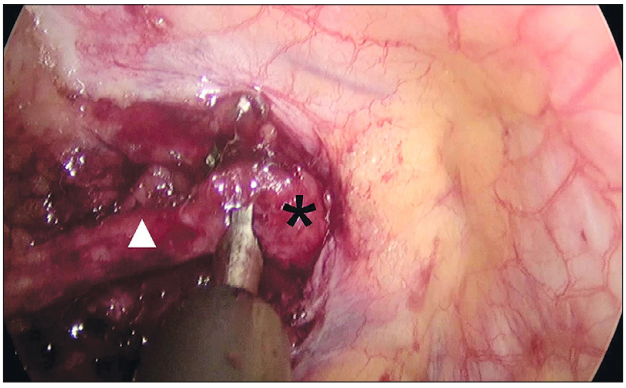

Fig. 3. Transecting the stellate ganglion (white arrowhead).

Treatment options and indications

Initial treatment for VA includes anti-arrhythmic drugs such as beta-blockers, non-dihydropyridine calcium channel blockers, amiodarone, propafenone, implantable cardioverter-defibrillator and catheter ablation. If these methods fail and a suboptimal outcome is achieved with VA recurrence and electrical storm, an acute treatment is required. In this acute setting, initial intubation and sedation of the patient with intravenous beta-blocker or anti-arrhythmic agent treatment are required. If not, other successful bedside treatment modalities could be utilised, such as thoracic epidural anaesthesia, percutaneous sonography or guided stellate ganglion block (10). These techniques have their limitations in the setting of antiplatelet and anticoagulant therapy. If catheter-based cardiac radiofrequency ablation fails repeatedly or is contraindicated, the surgical treatment remains the only viable and promising treatment option aside from heart transplantation (11). A more detailed description of the complex anti-arrhythmic therapy of VA is beyond the scope of this article.

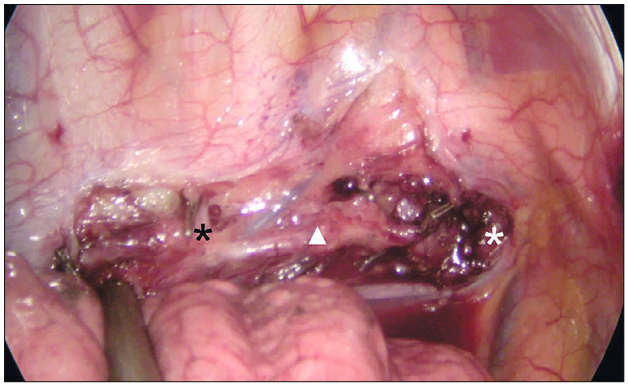

Fig. 1. Dissected and clipped sympathetic chain down to the level of T4 ganglion. (white star – transected stellate ganglion, white arrowhead – sympathetic chain, black star – *rami communicantes*).

Cardiac sympathetic denervation – surgical technique

In correlation with the latest published data, we are accustomed to the bilateral variant of thoracoscopic cardiac sympathetic denervation technique affecting both the left and right stellate ganglia and T2–T4 thoracic ganglia (12). The rationale not to perform just a unilateral left sided CSD is because many works demonstrated the importance of RSG in ventricular arrhythmia formation. Due to anatomic variability in the course of preganglionic sympathetic fibres, not all sympathetic connections to the heart may be severed, and this fact is emphasised when a solely unilateral approach is applied. The right cardiac RSG may hy-

pertrophy and extend nerve sprouts to the regions of previously resected LSG (13).

We specifically exploit our significant experience of endoscopic sympathetic block for hyperhidrosis and apply similar surgical steps during CSD. This modification is, to our knowledge, unique in the treatment of refractory VA.

When other treatment options fail, the patient who responded satisfactorily to percutaneous stellate ganglion block is routinely referred to a dedicated anaesthesiologist after the indication for CSD is made in cooperation with the arrhythmology department. If there are no contraindications to general anaesthesia and one-lung ventilation, the operation is conducted in a lateral decubitus position, and after deflation of the lung, we prefer to start in the left pleural cavity. Two 5-mm thoracoscopic or laparoscopic ports depending on the need for CO_2 insufflation are placed in the 3rd and 4th intercostal spaces in the anterior and posterior axillary lines. A 5-mm 30-degree thoracoscope is inserted, the stellate ganglion and sympathetic chain down to the T4 level are identified and dissected using a harmonic hook (Figs 1 and 2). This electric device converts high-frequency ultrasound energy into mechanical energy to cauterise tissues. This device prevents damage to neural structures, especially seen in Horner´s syndrome which is encountered when using electrocoagulation. In addition to the sympathetic trunk and ganglions, the *rami communicantes* are also dissected. In contrast with other papers where all of the above-mentioned neural structures are excised, we use the method of the sympathetic block with titanium clips applied to different levels of the *truncus sympathicus* and *rami communicantes* (Fig. 1). The lower half of the stellate ganglion is divided using the combination of harmonic hook and scissors (Fig. 3). A CH20 chest tube is placed in both pleural cavities using one of the original incisions and left there for the early post-operative period to evacuate any excess air or blood from the pleural cavity. After surgery, dedicated anaesthesiologists and staff observe the patient in the ICU.

Chest X-ray after surgery shows the levels of sympathectomy via the titanium clips.

Complications

Complications of CSD could be categorised into two groups (14). In the first group, there are complications directly related to the intervention on the sympathetic chain, such as Horner's syndrome, nocturnal anisocoria, facial anhidrosis and compensatory hyperhidrosis. The second group comprises complications associated with general anaesthesia and double-lumen intubation for one-lung ventilation and general complications related to video-assisted thoracoscopic surgery (VATS) such as pneumothorax, haemothorax, neuralgic post-incisional pain, and brachial plex injury related to patient positioning (15, 16). The incidence of the latter group is generally low due to the fact that the operation is usually carried out by a thoracic surgeon well-versed in minimally invasive techniques. Other complications such as Horner's syndrome could be reduced using proper countermeasures like the use of the harmonic hook and mechanical scissors while avoiding electrocoagulation. Some of them, including compensatory hyperhidrosis, cannot be effectively avoided due to the nature of the intervention and its targeted organ. Compensatory sweating on a different part of the body like the chest, back, or abdomen results as a consequence of sympathectomy based on negative afferent feedback (17). Its degree varies from mild through moderate to intense and could be perceived differently by individual patients. Patients experience dry hands immediately after surgery which is the usual goal of the endoscopic sympathetic block in hyperhidrosis patients. It requires moisturisers and this adverse effect of surgery together with compensatory sweating needs to be carefully and emphatically explained to the patients in detail before surgery.

Patient 1

A 34-year-old patient with confirmed type 2 muscular dystrophy with progressive heart failure with implanted ICD in primary prevention of sudden cardiac death. He manifested with an arrhythmic storm with multiple ICD shocks. Intravenous amiodarone and propafenone were ineffective. A catheter ablation of multiple ventricular tachycardias was performed, but a few weeks later, the patient experienced multiple shocks again due to a ventricular arrhythmic storm. Subsequently, a CSD was performed, and since the procedure, we observed only four shocks in the context of severe hypokalaemia due to diuretic agents. After ionogram correction, the patient was free of shocks. Afterwards, the patient underwent heart transplantation.

Patient 2

A 55-year-old patient with ischaemic cardiomyopathy after old STEMI (ST-elevated myocardial infarction) manifested as an arrhythmic storm with multiple ICD shocks. After initial unsuccessful treatment with anti-arrhythmic agents, a catheter ablation of multiple VT (ventricular tachycardia) sites was performed. After eight months of shock-free period, the patient manifested again with multiple appropriate ICD shocks due to VTs. All available anti-arrhythmic agents have had a minimal effect on the recurrence of shocks. After subsequent CSD, the patient has been free of shocks until now. He underwent heart transplantation.

Patient 3

A 39-year-old patient with recurrent syncopes due to *torsades de pointes*, rapid supraventricular tachycardia, and genetically confirmed long QT syndrome. After the initial implantation of the cardioverter/defibrillator, the patient experienced several inappropriate shocks due to rapid SVT (supraventricular tachycardia) with a heart rate over 250 bpm, despite maximum tolerated beta-blocker therapy. After two catheter ablations (pulmonary veins isolation, ablation of sinoatrial and atrioventricular node), the effect of ICD was still inappropriate. Finally, we performed CSD, and after the procedure, the patient reports higher tolerance of physical activity and is free of ICD shocks.

Tab. 1. Patient characteristics and results.

No	Age	Sex	Arrhythmia	Cardiomyopathy	ICD	Prior ablation	Stellate ggl. block	Transplant Waiting list	Shock during month prior to VATS	Technique	No of shocks after VATS	Shock-free post VATS period in weeks	Complication	Follow-up in weeks
1	34	M	VA,ES	dilated	yes	Yes	Yes	Yes	25	Bilateral VATS CSD	4 (hypo K+)	3 hypo K+11	None	14
2	55	M	VT,ES	dilated	Yes	Yes	Yes	yes	39	Bilateral VATS CSD	0	13	None	13
3	39	F	VT, AF, LQTS	no	yes	yes	yes	no	6	Bilateral VATS CSD	0	29	none	29

ICD – implantable cardioverter defibrillator, VATS – video-assisted thoracoscopic surgery, CSD – cardiac sympathetic denervation

Conclusion and discussion

Patients with refractory VT/VA and electrical storms are at high risk of sudden cardiac death. The latest retrospective single institutional studies indicate that if other treatment modalities such as pharmacological medication, ICD and catheter ablation fail to achieve optimal results, bilateral thoracoscopic cardiac denervation is a safe and feasible method with promising results. Most studies show an effectiveness of 70–80 % in the first month through one-year follow-up period after the procedure (18, 19).

In our limited experience, patients who underwent surgery showed a decreased number of ICD shocks or cessation thereof altogether with no periprocedural complications. In two of the patients, this minimally invasive procedure helped them bridge the time to receive heart transplantation because of the structural heart disease (Tab. 1). Despite its advantages, this treatment option is still underutilised, and published data are scarce. Prospective studies are required to establish its definitive position in refractory VT/VA treatment. A clear consensus on indication criteria is also needed; in fact, close cooperation with cardiologists is vital to the proper treatment of patients and referrals to cardiothoracic surgery units. According to the published data, the pathological conditions eligible to CSD include refractory ventricular arrhythmias secondary to ischaemia, monomorphic VT originated from a right ventricular scar (arrhythmogenic right ventricular cardiomyopathy), a structurally normal heart, nonischaemic cardiomyopathy without a scar, polymorphic VT, hereditary arrhythmias, channelopathies, and arrhythmias due to long QT syndrome (LQTS) (20–25).

One of the limitations of CSD has been the inability to determine the immediate success of the procedure. Inadequate denervation may be revealed only when recurrence of VA occurs. However, the response to VATS CSD can be evaluated by comparing the number of pre-surgery to post-operative ICD shocks. Another limitation of CSD arises from the anatomic variability of nerve pathways. Inevitable complications such as compensatory sweating need to be addressed and adequately explained to the patient, emphasising the benefit of the procedure. Its advantage is the minimally invasive nature of this technique with low periprocedural morbidity. To improve the results of CSD, the means of proper cardiac-specific sympathetic neuron targeting are needed. Optimising the timing of CSD in the treatment regime could also help to improve the results. More accurate and immediate assessment of denervation and proper patient selection would also aid in enhancing the "popularisation" of this modality. For an adequate outcome, it is necessary to undertake multidisciplinary approach involving experienced cardiologists, anaesthesiologists, and thoracic surgeons, and therefore, the centralisation to high-volume centres seems natural.

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