INTRODUCTION

An electrical storm (ES) in ischaemic heart disease usually manifest as recurrent ventricular arrhythmias that are resistant to the antiarrhythmic drugs.\(^1\) We report an incident of ES in a patient, which was treated with left stellate ganglion block (LSGB) under ultrasound guidance followed by left cardiac sympathetic denervation (LCSD) under general anaesthesia. We also describe the anaesthetic implications of the LCSD.

CASE REPORT

A 52-year-old male, weighing 55 kg, received stents in left anterior descending artery (LAD) and first diagonal artery and a permanent pacemaker (VDD) following anteroseptal myocardial infarction (MI) with complete heart block 2 years ago. Postoperative course was complicated with recurrent ventricular tachycardia (VT) and syncopal attacks requiring hospitalization and DC cardioversion on four occasions. The pacemaker was found to be properly functioning during this period. He was readmitted 5 days ago for electrophysiological studies and radiofrequency ablation for VT. Cardiology evaluation revealed pulse rate of 70/min and systemic blood pressure (BP) of 100/70 mmHg. Findings on transthoracic echocardiography were a dilated left ventricle with a very low ejection fraction (20%), akesis of LAD territory and grade-1 mitral regurgitation. Preoperative medication consisted of oral amiodarone 300 mg and sustained release metoprolol 50 mg once and twice a day respectively. During electrophysiological studies, VT could be easily induced, and the VT circuits were mapped and ablated at the anterobasal left ventricle.
After an arrhythmia-free interval of 24 hours, he started having repeat episodes of monomorphic VT, degenerating into polymorphic VT and VF, requiring frequent cardioversions [Figure 1].

Hypokalemia, hypomagnesemia and hypothyroidism were excluded as triggering factors for arrhythmias on laboratory investigations. Severe hypotension and episodes of transient loss of consciousness warranted ventilatory therapy and inotropic support with epinephrine 0.1 mcg/kg/min. The trachea was intubated after administration of 200 mcg of fentanyl, 3 mg midazolam, 30 mg propofol and muscle relaxation with vecuronium 10 mg. Sedation was maintained using infusions of midazolam 1 mg/hour and fentanyl 60 mcg/hour. As the VT was resistant to treatment with magnesium, amiodarone and lidocaine LSGB was performed under ultrasound guidance using a 7.5 MHz probe (Site-Rite ultrasound system, Bard Access, Inc., USA). A 23-G 4-cm long needle was advanced through the prevertebral fascia till its tip was placed in the longus colli muscle. Injection of 6-ml 0.25% Bupivacaine solution resulted in bulging of the longus colli compartment and caudad and cephalad spread, which was verified on ultrasound. Warming of left upper limb and left Horner’s syndrome confirmed the success of LSGB. Patient remaining arrhythmia-free for 2 hours immediately after the blockade and incidents of VT were dramatically reduced subsequently for 12 hours.

Considering the benefits of LSGB toward pacifying the ES, we decided to perform LCSD under general anaesthesia via supraclavicular surgical approach. Placing high thoracic epidural catheter and video-assisted thoracoscopy (VAT)-guided ablation of left upper thoracic sympathetic ganglia were other options, which, however, were considered inappropriate given the unstable hemodynamic condition of patient and deranged left ventricular function. Upon arrival in the operation suite, his heart rate and systemic blood pressure were 78 bpm and 90/68 mmHg, respectively. External defibrillator paddles were placed around chest. Monitoring essentially consisted of electrocardiogram (leads II and V5) and invasive arterial blood pressure. Since the endotracheal tube was in situ, anaesthesia was deepened with bolus administration of fentanyl 200 mcg, midazolam 2 mg, propofol 25 mg and vecuronium 6 mg and maintained with sevofturane adjusting concentration to maintain BIS between 50 and 60. Cervicothoracic sympathectomy was performed through neck excising left stellate ganglion and second thoracic ganglion with sympathetic chain. No further deterioration occurred in the intraoperative period and the patient was transferred back to the ICU for elective ventilation. The VT load significantly abated in the immediate postoperative period. Trachea was extubated 8 hours after surgery and inotropic support was tapered off over next 24 hours. He was discharged in a hemodynamically stable condition and remained symptom-free without any incident of VT for past 8 months.

DISCUSSION

An ES is defined as recurrent hemodynamically unstable VT and/or ventricular fibrillation (VF), thrice or more in 24 hours, requiring intervention of the defibrillator (anti-tachycardia pacing or shock). Ongoing myocardial ischaemia or reentrant circuits in an old infarct scar are considered causative factors for ES, which are associated with augmented sympathetic activity and increased propensity for ventricular arrhythmias. Recurrent VT or VF may result in left ventricular systolic dysfunction and myocardial injury exacerbating heart failure.

Identifying and reversing the causative factors is important during treatment. Specific precipitants include acute myocardial ischaemia, heart failure, hypokalemia, hypomagnesemia, arrhythmogenic drug therapy, hyperthyroidism, infection and fever. Treatment options for ES include correcting the causative factors, providing hemodynamic support, instituting antiarrhythmic therapy, placing implantable cardioverter-defibrillator (ICD), achieving...
cardiac sympathetic denervation and interrupting the reentrant arrhythmia pathways using radiofrequency ablation. Nademanee et al.,[3] reported that cardiac sympathetic blockade is superior to the antiarrhythmic therapy in treating ES and have advocated LSGB for reducing the sympathetic surge. There are a few publications on efficacy of the LSGB[6,7] and thoracic epidural anaesthesia (TEA)[8,9] in lowering the incidents of ventricular arrhythmias during ES. These therapeutic approaches reduce adrenergic tone that is most likely responsible for the reported efficacy. LSGB also reduces the risk of cardiac arrhythmias by shortening the QT interval.[10] The antiarrhythmic effect of LSGB was reported in patients with MI[11] and prolonged QT interval syndrome.[12]

LCGB provides distinct but transient abolition of ES which can be utilized as the therapeutic test for sympathetic denervation. LCSD interrupts the major source of norepinephrine released in the heart, increases the threshold for VF and ventricular refractoriness.[13] LCSD probably results in some kind of remodeling in the cardiac sympathetic innervations.[14] Wilde et al.,[13] described that surgical LCSD in three young adults with polymorphic ventricular tachycardia resulted in postoperative cessation of symptoms. ICD is another treatment option, which, however, may worsen the arrhythmias in catecholaminergic VT. Repeated shocks delivered through the ICD may aggravate sympathetic stimulation, ventricular arrhythmias and initiate a series of more shocks and ES.[13,15,16] The LCSD may complement the use of an ICD, because the denervation markedly decreases the catecholaminergic load on the heart, which may facilitate smooth functioning of the ICD in interrupting VF and restoring the sinus rhythm.

Performing LSGB under the ultrasound guidance is beneficial in many respects over the blind landmark technique. Volume of local anaesthetic solution is an important concern during the cervicothoracic ganglion blockade against the likelihood of toxicity. Wulf et al.[17] observed toxic plasma levels of bupivacaine in 30% of patients given injection of 10 ml of 0.5% bupivacaine during SGB. A large volume of local anaesthetic, as much as 20 ml may be required to achieve desired blockade during blind injection[18] as uncertainty exist over the placement of needle tip in the vicinity of the stellate ganglion. On the contrary, 5-6 ml solution may be sufficient to block the ganglia as it can be injected precisely in the longus colli muscle under the ultrasound guidance. Success rate of the blockade technique is enhanced by visualizing distention of the longus colli compartmental space under the pretracheal fascia, directly under the ultrasound imaging. Blind landmark technique of LSGB is fraught with complications like accidental injection into the vertebral artery and injury to superior thyroid artery leading to hematoma formation in 25% of patients.[19] Esophageal injury and pneumothorax are other complications which may be averted by ultrasonographic assistance.[20]

Sympathetic denervation in the presence of ES poses a challenge to the anaesthesiologist, as the stress of surgery may further derange the cardiac function and aggravate the arrhythmia storm. Although high TEA may lower the cardiac sympathetic tone, the unstable hemodynamic condition precluded us from inserting the catheter as turning the patient in lateral decubitus or sitting position would have worsened the situation. Surgical LCSD involves excision of cervicothoracic and upper thoracic ganglia along with the intervening sympathetic chain, which may be accessed via video-assisted thoracoscopy (VAT) or through a supraclavicular incision following an extrapleural approach without performing thoracotomy. VAT sympathectomy demands changing over the endotracheal tube with a double lumen endobronchial tube for one-lung ventilation and turning the patient in right lateral decubitus, which would have deteriorated hemodynamic and respiratory parameters in our patient. Hence we preferred to perform the LCSD via supraclavicular approach. Avoiding sympathetic stimulation arising from light anaesthesia, tachycardia, hypothermia, hypoxaemia and hypercapnia is of vital importance while anaesthetizing these patients. Electrolyte imbalances like hypokalemia, hypomagnesaemia, and hypocalcaemia can contribute to delayed myocardial repolarization and should be treated promptly. Short-acting anaesthetics such as propofol and benzodiazepines have been found to successfully convert and suppress occurrence of VT[21] during ES. Propofol and inhalational anaesthetics like sevoflurane and isoflurane are accepted well by these patients.[22]

**CONCLUSION**

ES associated with catecholaminergic surge may be resistant to the antiarrhythmic drugs. LSGB followed by surgical LCSD is an effective treatment modality to reduce the incidents of malignant ventricular arrhythmias. LSGB may serve a simple bedside tool to identify the patients who are likely to respond
to the more definite but more invasive modality of LCSD. The ultrasonography during LSGB enhances the success rate of the technique, reduces the quantity of local anaesthetic required to produce desired effects and prevents technical complications. Supraclavicular surgical access to the upper thoracic sympathetic chain obviates the necessity for one lung ventilation and lateral decubitus during surgery in a hemodynamically unstable patient. Sympathectomy can be performed under general anaesthesia essentially avoiding sympathetic stimulation.

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