Successful neural modulation of bedside modified thoracic epidural anesthesia for ventricular tachycardia electrical storm

Ki-Woon Kang

Division of Cardiology, Chung-Ang University Hospital, Chung-Ang University College of Medicine, Seoul, Korea

Ventricular tachycardia (VT)/ventricular fibrillation (VF) storm can be hemodynamically compromising and life-threatening. Management of medically refractory VT/VF storm is challenging in the intensive care unit. A 38-year-old male patient was diagnosed with non-ischemic heart failure and acute kidney injury with documented frequent premature ventricular contraction with QT prolongation after recurrent VT/VF. Even though the patient was intubated with sedatives and had taken more than two anti-arrhythmic drugs with external recurrent defibrillation at bedside, the electrical storm persisted for several hours. However, medically refractory VT/VF storm can be successfully and rapidly terminated with a modified thoracic epidural anesthesia at bedside. This case demonstrates that a bedside thoracic epidural anesthesia can be an effective non-pharmacological option to treat medically refractory VT/VF storm in the intensive care unit.

Key Words: epidural anesthesia; intensive care unit; ventricular tachycardia

Ventricular tachycardia (VT)/ventricular fibrillation (VF) electrical storm is occurrence of three or more separate VT/VF episodes in a 24-hour period. The guideline recommends neural modulation for sympathetic hyperexcitation of the thoracic stellate ganglion in patients with VT/VF storm in whom beta blockers, other antiarrhythmic medications, and catheter ablation are ineffective, not tolerated, or not possible [1]. Therefore, a medically refractory VT/VF electrical storm at bedside is challenging in the intensive care unit.

CASE REPORT

A 38-year-old patient was referred to the intensive care unit with a diagnosis of frequent premature ventricular complex (PVC) and VT/VF storm underlying heart failure with reduced ejection fraction (39%). The etiology of heart failure was non-ischemic and arrhythmia-induced cardiomyopathy due to normal coronary angiogram. The normal arterial blood gas analysis and electrolyte were normally maintained with continuous renal replacement therapy due to acute renal failure. PVCs and non-sustained VT frequently appeared and degenerated to sustained VT/VF. Restored sinus rhythm presented a prolonged QT interval.
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Figure 1. (A) Rhythm strip shows a prolonged QT interval with a giant T-wave inversion (blue dotted line) and sudden initiation of ventricular tachycardia. Black arrow indicates first premature ventricular beat inducing ventricular tachycardia. (B) Rhythm strip shows sustained ventricular fibrillation degeneration in the setting of a long QT interval. (C) Rhythm strip shows that the incessant ventricular tachycardia/fibrillation was promptly terminated (asterisk) and restored to a sinus rhythm (blue dotted line) after the thoracic epidural anesthesia. ECG: electrocardiogram.

with giant T-wave inversion (Figure 1), and continuous electrocardiogram monitoring showed recurrent short-episodes of Torsade de pointes (Tdp). The patient developed electrical storm after defibrillation and required sedation with intubation (midazolam continuous infusion, 7.2 mg/hr, deep station state, Richmond Agitation-Sedation Scale 4), and intravascular lidocaine and amiodarone with magnesium sulfate supplement. Additional isoproterenol infusion or temporary pacing was not effective due to more frequent stimulation for occurrence of non-sustained VT. Repeated direct current defibrillation succeeded in converting short episodes of TdP to sinus rhythm, which finally recurred to frequent PVC and non-sustained VT. Based on the recent guideline [1] for patients with VT/VF storm in whom a beta blocker, other antiarrhythmic medications, and catheter ablation are not tolerated or not possible, sympathetic neural modulation might be reasonable (class IIb). We applied a bedside modified thoracic epidural anesthesia in which an 18-G Tuohy needle was inserted in the lumbar spine using a standard loss of resistance technique to place the epidural catheter under aseptic precautions in the lateral decubitus position. The tip of the epidural catheter with an intraluminal 0.014-inch-diameter intervention wire (Run-through, 180 cm length, 3 cm tip radiopacity) was advanced. The intervention wire was retracted between T1 and T2 guided by a portable X-ray, and 5 ml of 0.25% bupivacaine with 3 ml of 2% lidocaine was injected (Figure 2) [2]. The incessant VT/VF episode was promptly terminated and did not recur, with improved QT interval and T-wave inversion (Figure 3); no procedural complications were observed.

DISCUSSION

VT/VF storm can be hemodynamically unstable and subsequently life-threatening. Its bedside management is challenging due to the pathogenesis of sympathetic hyperexcitation with a vicious cycle regardless of ischemic or non-ischemic etiology [1]. The incidence of Tdp associated with intravenous amiodarone was lower than 1.5% [3], but advanced heart failure with sympathetic hyperexcitation might influence the risk of increased transmural dispersion of repolarization, resulting in marked QT prolongation with T-wave inversion. Further, additional amiodarone administration produces preferential prolongation of action potential duration of the myocardium so that the QT interval is prolonged, increasing the risk of TdP occurrence [4]. A systematic review supports invasive neural modulation on the thoracic stellate ganglion as an effective treatment for autonomic dysregulation in the pathogenesis of VT/VF storm [5,6]. A thoracic epidural anesthesia could be considered as an effective option to increase the threshold and lengthen ventricular repolarization and effective refractory
Figure 2. (A) Intubation tube (black arrow), right jugular vein sheath and defibrillation patch (asterisk) and linear thoracic epidural catheter (white arrows). (B) Anatomical position of 18-G Tuohy needle with loss of resistance technique.

Figure 3. (A) A 12-lead electrocardiogram (ECG) shows a prolonged QT interval with a giant T-wave inversion before initiation of ventricular tachycardia at the period of the blue dotted line in Figure 1A. (B) A 12-lead ECG shows normalized QT prolongation with T-wave inversion after the thoracic epidural anesthesia at the period of the blue dotted line in Figure 1C. aVR: augmented vector right; aVL: augmented vector left; aVF: augmented vector foot.
periods by shortening the activation recovery interval and spatial heterogeneity of the repolarization caused, as well as to decrease the ventricular arrhythmia burden [1,2,7]. Recently, the benefits of thoracic epidural anesthesia have been reported to be its rapid action and minimal systemic complications compared with catheter ablation and surgical sympathectomy through local anesthetic directly on the stellate ganglion and sympathetic chain, which results in almost immediate sympatholysis [8-10]. Potential procedural complications from an epidural anesthesia (post-operative neurologic deficits, epidural hematoma, post-dural puncture headache, and systemic local anesthetic toxicity) are rare [11]. This case report supports an urgent bedside modified thoracic epidural anesthesia as effective therapy for neural modulation of sympathetic hyperexcitation in a medically refractory VT/VF storm.

CONFLICT OF INTEREST

No potential conflict of interest relevant to this article was reported.

ORCID

Ki-Woon Kang https://orcid.org/0000-0002-1361-0022

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