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CASE REPORT

Anesthetic Management in Cardiac Sympathetic Denervation

Adhe Kurniawan[✉*], Reza Widianto Sudjud[*]

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Authors' affiliations:
[*] Department Anesthesiology and Intensive Therapy, Dr. Hasan Sadikin General Hospital, Bandung
✉Correspondence:
hasanalbashri@gmail.com

ABSTRACT

Background : This case report discusses anesthetic management of cardiac sympathetic denervation (CSD), with a particular focus on strategies for maintaining optimal perioperative hemodynamic stability.

Case Illustration : A 59-year-old woman diagnosed with dilated cardiomyopathy accompanied by impaired ejection fraction was treated for refractory ventricular tachycardia. She experienced recurrent ventricular tachycardia despite having undergone radiofrequency CA and receiving pharmacologic agents such as beta-blockers and antiarrhythmic drugs. She underwent CSD procedure through video-assisted thoracoscopic surgery (VATS) approach under general anesthesia. In addition to standard monitoring and invasive blood pressure monitoring, the preparation of an external defibrillator, vasopressors, and inotropic agents were necessary prior to the induction of anesthesia. Intravenous induction agents were administered in small initial doses and increased gradually according to the response of the patient. CSD was performed through a left side sympathetic ganglionectomy using VATS approach. During CSD procedure, patient was placed in supine position to reduce the risk of hemodynamic instability associated with position change to right lateral decubitus and to facilitate cardiopulmonary resuscitation and defibrillation if ventricular tachycardia and/or ventricular fibrillation occur perioperatively. The patient was extubated in the operating room and transferred to ICU safely.

Conclusion: Anesthesiologists must determine the hemodynamic targets to be achieved before inducing patients with dilated cardiomyopathy, so that several things must be appointed including the patient clinical status and the degree of cardiac function, the appropriate monitoring devices and anesthetic agents, and other resources which required to activate the ACLS protocol to maximize perioperative survival.

Keywords: Anesthesia; Cardiac Sympathetic Denervation; Dilated Cardiomyopathy; Ventricular Tachycardia.

INTRODUCTION

Dilated cardiomyopathy (DCM) is characterized by left ventricular or biventricular enlargement and impairment of systolic function, with or without congestive heart failure. Anesthetic management of patients with DCM undergoing noncardiac surgery is challenging and is associated with high mortality. The majority of DCM cases were initially thought as idiopathic (66%). However, increasing evidence has shown that DCM has a familial basis.

Dilated cardiomyopathy mainly manifests as reduced ejection fraction (EF) and cardiac output (CO). The decrease in forward blood flow leads to an increase in ventricular end-diastolic volume, ventricular filling pressure, and eventually leads to ventricular enlargement to maintain CO. DCM is often accompanied by malignant arrhythmias, heart failure, mitral or tricuspid regurgitation, and sudden death. Although the 5-year mortality rate has decreased significantly, sudden death from DCM accounted for 30% of all deaths.[1]

Malignant arrhythmias include ventricular tachycardia (VT), with 10% occurs in those with structurally normal heart and these are called as idiopathic VTs, and the rest 90% occurs in patients with structural heart disease. Incessant VT is defined as continuous sustained VT during several hours, which recurs promptly despite repeated intervention for termination. VT storm is considered as ≥ 3 separate episodes of sustained VT within 24 hours, each requiring termination by an intervention.[2]

Ventricular Tachycardia storm refers to ventricular arrhythmias refractory to medical treatment, for which the need for electrical therapy may range from twice in a 24-hour period to nearly continuous shocks. Such arrhythmias classically are treated with a combination of antiarrhythmic drugs, defibrillation, and/or rapid pacing. Class I antiarrhythmics often fail, and amiodarone may take days to achieve sufficient rhythm control. VT storm refractory to medical and electrical therapies has been treated successfully via catheter ablation, although the failure rate of this approach remains high, thus necessitating alternative treatment. Cardiac sympathetic denervation (CSD) has been introduced as a definitive surgical approach to ameliorate sympathetically mediated VT in patients refractory to conventional therapies, and its use is increasingly gaining

acceptance. Patients who are candidates for this approach universally present on both an emergency basis and often moribund cardiac state.[3]

Cardiac sympathetic denervation with video-assisted thoracoscopic surgery (VATS) approach was first described in 2000 and nowadays has become a therapeutic option for refractory ventricular tachycardia, this procedure showing a 70-80% effectiveness to reduce burden due to ventricular tachycardia and improve quality of life of patients within the first year following the surgery.[4] Cardiac sympathetic denervation is the last resort for the management of patients with ventricular arrhythmias. Candidates for this procedure are often in unfavorable clinical conditions and have limited physiological reserves. The anesthesiologist must plan the perioperative management meticulously, prior to induction of general anesthesia so that avoid terrifying condition like life-threatening ventricular arrhythmias.

CASE ILLUSTRATION

A 59-year-old woman arrived to the emergency department with complaint of recurrent chest-pounding since 3 weeks ago, accompanied with weakness and near-syncope. According to information from doctor who treated the patient previously, patient had been diagnosed with dilated cardiomyopathy and refractory ventricular tachycardia. Patient had received pharmacologic therapy with intravenous infusion amiodarone 0.5 mg/hour in the previous hospital. At the emergency department, the patient's condition deteriorated due to recurrent ventricular tachycardia, accompanied by hemodynamic instability and a decrease in level of consciousness (Figure 1). Patients had received several cardioversion and pharmacological therapy. After hemodynamic stability was achieved, she was transferred to the high care unit.

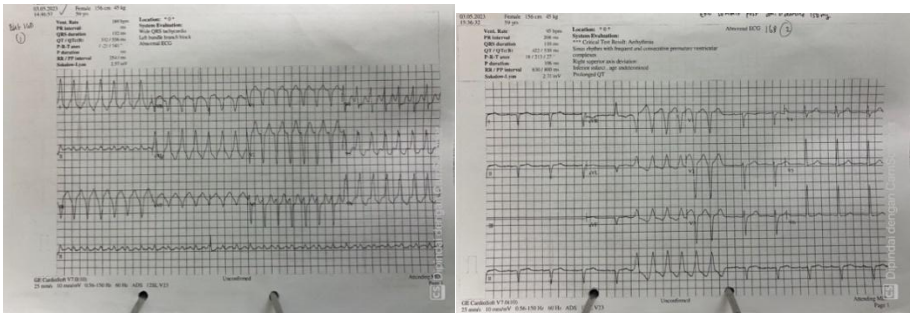

Figure 1. Electrocardiography examinations in the emergency department demonstrated ventricular tachycardia (left), which subsided after the administration of amiodarone (right).

Echocardiographic examination revealed a dilated left ventricular (LV), eccentric left ventricular hypertrophy (LVH), reduced LV systolic function (LVEF 33%) with regional wall motion abnormalities and regional RV wall akinesia. Coronary angiography examination showed normal coronary arteries. Electrocardiography examination demonstrated sinus rhythm with prolonged QT interval (QTc 524 ms) (Figure 2). During treatment at the high care unit, the patient experienced recurrent ventricular tachycardia despite having undergone radiofrequency catheter ablation, receiving beta-blockers, and antiarrhythmic drugs. Because patient refused implantable cardioverter defibrillator (ICD), the patient was scheduled to undergo CSD through video-assisted thoracoscopic surgery (VATS) approach.

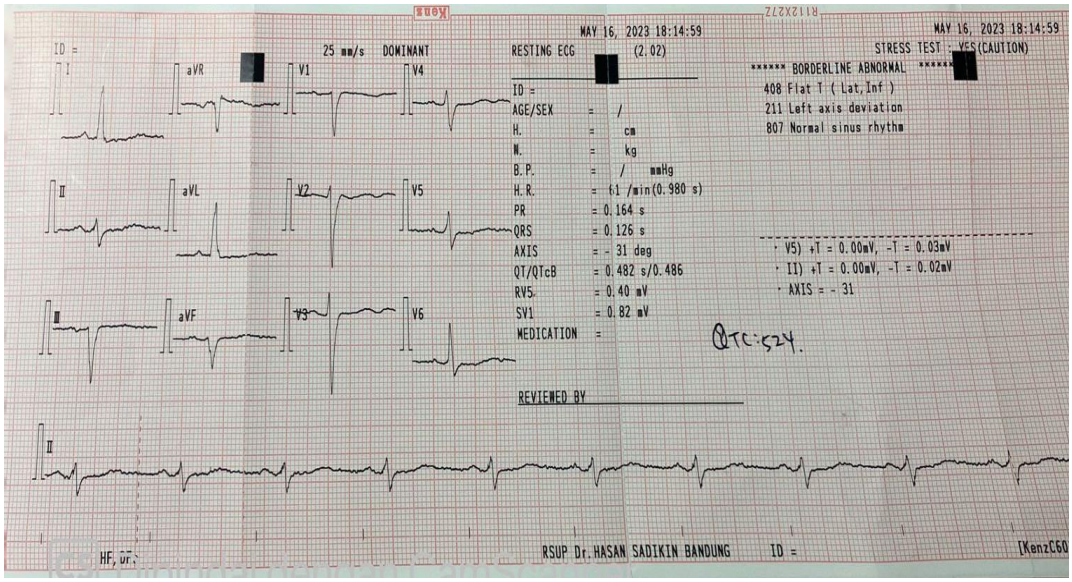

Figure 2. Electrocardiography examination in the high care unit one day before surgery, have showed prolonged QT interval (QTc 524 ms).

The patient complained of shortness of breath and felt more comfortable with the fowler's position. Patient was alert with noninvasive blood pressure measurement 106/65 mmHg, regular pulse of 72 beats per minute, respiratory rate 24 times per minute, peripheral oxygen saturation 98% with oxygen supplementation 3 liters per minute, and body weight of 60 kg. Physical examination revealed crackles on lung bases. On laboratory examination, the results of hematology, kidney function, liver function, and electrolytes were within normal limits. On the thyroid scan, the results of fT4

and TSHs were also within normal limits. Patients had received medications such as bisoprolol 10 mg qd, sacubitril valsartan 50 mg bid, spironolactone 25 mg qd, empaglifozin 12.5 mg qd, and amiodarone 200 mg bid. The patient already had peripheral venous access installed with IV catheter number 18, an additional peripheral venous access with large bore catheter was inserted to anticipate the use of inotropic agent.

Standard monitoring and cannulation of the radial artery for invasive blood pressure monitoring were carried out. An external defibrillator, vasopressors, and inotropic agents should be prepared before induction of anesthesia. Personnel capable of initiating ACLS protocols were present to anticipate ventricular tachycardia and/or ventricular fibrillation, thereby maximizing perioperative survival.

Induction of general anesthesia was performed in a 30 degree head up position and oxygen supplementation 3 liters per minute. Intravenous induction agents were administered in small initial doses and increased gradually according to the response of the patient, with total doses of fentanyl 200 mcg and midazolam 10 mg. Atracurium 30 mg was administered as muscle relaxant to facilitate smooth intubation. Lidocaine 90 mg was also given intravenously to blunt sympathetic response during laryngoscopy and intubation. Patient was intubated with a 37 Fr left side double lumen tube (DLT) and maintenance anesthesia with sevoflurane 2 vol%. A foley catheter was inserted after induction of general anesthesia. CSD was performed with left side sympathetic ganglionectomy using VATS approach in supine position. During the intraoperative period, left lung down was performed to facilitate trocar insertion into the thoracic cavity. Hemodynamic instability was observed and corrected with intravenous dobutamine 5 mcg/kg/minute. The procedure was lasted for 90 minutes with minimal bleeding. After the procedure was finished, a chest tube was inserted in the left hemithorax.

The patient was extubated in the operating room and monitored for consciousness and hemodynamic status in the recovery room. Intravenous analgesics administered included with morphine 10 mcg/kg/hour and paracetamol 1 gram/6 hours. Administration of inotropic agents (dobutamine 5 mcg/kg/minute) was maintained until the patient was

transferred to the intensive care unit. Patient was discharged in a hemodynamically stable condition after four days and remained symptom-free without any incidents of ventricular tachycardia.

DISCUSSION

The clinical incidence of dilated cardiomyopathy (DCM) is 2.45 cases per 100,000 population per year. Anesthetic management of patients with cardiomyopathy with reduced systolic function is challenging and may be associated with high mortality.[1] The prognosis of DCM patients depends upon the degree of systolic dysfunction of left ventricle, with threshold limit of 35% for high risk of sudden cardiac death.[5]

DCM is a primary myocardial disease of varied etiological causes. It is characterized by left ventricular or biventricular dilatation and impaired ventricular contractility. The term DCM is generally used to designate an idiopathic process, in the absence of long-standing hypertension, toxin exposure, or chronic alcoholism (secondary dilated cardiomyopathy). Malignant arrhythmias are the most common cause of death in DCM. Anesthetic management of these patients is quite challenging, because of the associated global ventricular dysfunction and the predisposition of these patients to malignant arrhythmias perioperatively, both of which are aggravated by the myocardial depressant effect of anesthetic drugs as well as surgical stress.[1]

Minimally invasive cardiac sympathetic denervation (CSD) by a video-assisted approach is treatment option for patients with incessant ventricular tachycardia. After induction of general anesthesia, a left-sided double-lumen endotracheal tube is placed for lung isolation, and the patient is positioned in right lateral decubitus position. If an automated external defibrillator is used, the pads should be placed to allow intraoperative use when needed.[6] In this case report, the patient was positioned supine during the CSD procedure to prevent hemodynamic instability and to facilitate cardiopulmonary resuscitation in the event of intraoperative ventricular tachycardia and/or ventricular fibrillation. The pads of external defibrillator were not applied to the patient's chest prior to induction of anesthesia because the automated external defibrillator was not available in

the operating theatre. Instead, the external defibrillator device was placed near the patient's environment, including during postoperative period in the intensive care unit.

When treatment with medications and catheter ablation fails in patients with refractory VT, surgical cardiac sympathetic denervation by bilateral or unilateral stellate ganglionectomy is preferred. These patients have comorbidities that make anesthesia care more challenging, requiring perioperative reprogramming of defibrillators and pacemakers if they used implantable devices, inotropic and/or vasopressor support, and preparation for the management of intraoperative ventricular arrhythmias. Although bilateral sympathectomy has shown to be better than unilateral sympathectomy, left-sided sympathetic denervation has also been shown to be more effective than right-sided denervation. A chest tube is routinely placed because postoperative effusions are prevalent in these patients due to heart failure.[6]

Because sympathetic innervation of the heart has been shown to play a key role in production of fatal arrhythmias, surgical sympathectomy has increasingly gained popularity as a treatment modality. Even so, this procedure is often considered only as a final option after a patient has failed medical treatment and catheter-based ablation.[3] Usually, cardiomyopathy and heart failure are followed by excess of sympathetic output and parasympathetic withdrawal, which contribute to ventricular arrhythmias. Therefore, autonomic modulation is increasingly being employed as a strategy to treat refractory tachyarrhythmia. CSD interrupts in the release of norepinephrine in the heart, increases the ventricular fibrillation threshold, probably results in some kind of remodeling in the cardiac sympathetic innervation, and does not impair cardiac contractility.

Bilateral denervation approach is as effective as the left side approach alone. Hypoxemia, hypercapnia, hypocapnia, acidosis, and superficial anesthesia should be avoided as these conditions affect the repolarization of the cardiac myocyte and increase the sympathetic tone, precipitating arrhythmias. During CSD, both single-lumen and double-lumen endotracheal tubes can be used, with or without intrathoracic CO_2 insufflation.[4]

Ventricular arrhythmias may be due to varying etiologies. However, it is known that ventricular arrhythmias are generally sensitive to, and can be triggered by, sympathetic stimulation. The left cardiac sympathetic system is recognized as predominantly regulating arrhythmogenic potential, whereas the right-sided system primarily regulates heart rate. Left sympathectomy is, therefore, usually the initial surgery of choice, although staged or concurrent right-sided sympathectomy also can be performed when the etiology of the ventricular arrhythmia is deemed to be more complex or particularly sensitive to sympathetic stimulation. The use of surgical sympathectomy for treatment of VT due to prolonged QT syndrome as well as catecholaminergic polymorphic ventricular tachycardia are mediated via decreased intracardiac norepinephrine release.[3]

Because the patients presented for this surgery were in a moribund cardiac state, all received arterial catheters to facilitate hemodynamic monitoring and management, as well as frequent blood sampling for blood gas analysis. Fluid responsiveness should be identified before starting volume expansion to treat hemodynamic instability. Fluid responsiveness can be estimated from plethysmographic waveform which derive from invasive blood pressure monitoring. The plethysmographic waveform can depict respiratory variation of arterial pulse pressure (PPV) and stroke volume (SVV) to positive-pressure ventilation. Dynamic monitoring approaches predict fluid responsiveness, thereby mitigating the risks of excessive fluid administration. Dynamic measures guiding therapy in patients with heart disease leads to less fluid administration with superior outcomes.[7] In this case report, the patient was monitored with standard monitoring devices as described by the American Society of Anesthesiologists with the addition of invasive blood pressure monitoring and end tidal CO_2.

The patients may require inotropic and vasopressor support, making an advanced understanding of the indications and use of these drugs crucial for optimal perioperative care. Furthermore, the ability and willingness to initiate ACLS protocols are absolutely necessary to maximize perioperative survival in these patients. Perioperative hemodynamic data were gathered including invasive blood pressure in the

24 hours before surgery, intraoperatively before sympathectomy and after sympathectomy, and in the first 24 hours after surgery, to assess hemodynamic effects of sympathectomy.[3] In this case report, perioperative invasive blood pressure and heart rate measurements are presented in the following table (Figure 3).

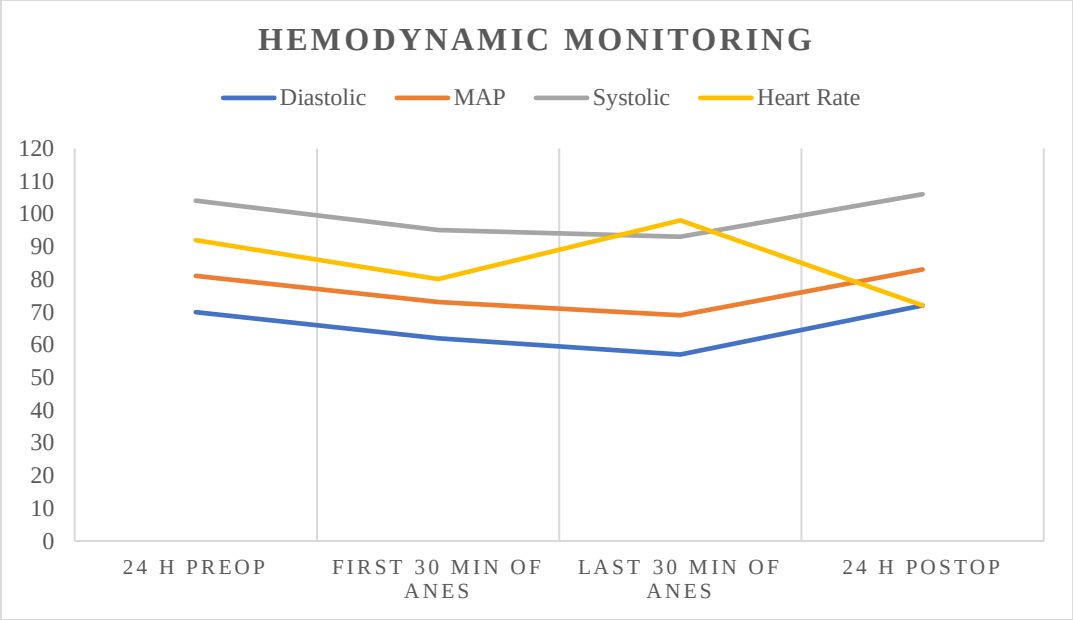

Figure 3. Hemodynamic Monitoring. The table shows the results of perioperative invasive blood pressure and heart rate measurements including 24 hours preoperatively, 30 minutes early after induction of anesthesia which shows hemodynamic before CSD, last 30 minutes during anesthesia shows hemodynamic after CSD, and 24 hours postoperatively.

The goals of anesthetic management for DCM are the maintenance of myocardial contractility by carefully titrating the anesthetic drugs, maintaining normovolemia, avoiding overdose of drugs during induction as the circulation time is slow, avoiding increase in ventricular afterload.[1] Other authors indicate that the anesthetic management also includes the maintenance of normal diastolic blood pressure to ensure coronary perfusion, maintenance of preload and preventing fluid overload, and avoidance of arrhythmias (tachycardia). When general anesthetic is chosen, it should have minimal inhibition on cardiovascular function according to the status of patients, and the minimal dose should be used to achieve the desired effect. Induction of general anesthesia should be administered with small doses, and increased gradually according to the response of the patients.[1]

Fentanyl and etomidate were preferred for patients with significantly

depressed ejection fraction (EF). Neuromuscular blockade was maintained by administration of a nondepolarizing agent (rocuronium, vecuronium, or cisatracurium).[3] Propofol is a commonly used intravenous anesthesia drug. Propofol can reduce LV preload and afterload, induce myocardial depression, and impair early-diastolic left ventricular filling, but this effect could be reversed by inotropic drugs. Midazolam is a commonly used sedative drug because it does not induce myocardial depression or vasodilation.[1]

The maintenance of anesthetic depth is important to prevent an increase in afterload. Potent inhaled anesthetic agents (0.7-1.3 minimum alveolar concentration) with a small dose (2-3 μg/kg) of fentanyl may be safely used because it did not decrease myocardial contractility. Opioids (fentanyl and sufentanil) have minimal side effects on cardiac function. If inotropic agents are required, dobutamine and low dose epinephrine can be used. Those agents can improve the stroke volume, while at the same time they can increase the cardiac work and the oxygen consumption. β-adrenergic agonists are the optimal vasoactive agents for DCM.[1]

In studies comparing anesthesia induction regimens, including the propofol ketamin regimen and the midazolam fentanyl regimen, the results showed that significant prolongation in QTc was detected using Bazett's formula in patients administered the propofol and ketamin combination for anesthesia induction. As propofol does not have a negative effect on QTc and even shortens it, the cause of this prolongation is likely ketamine. Hemodynamic parameters as an indicator of the sympathomimetic effect were significantly higher in propofol and ketamin combination. On the other hand, midazolam does not affect QTc or electrical distribution and it can be safely used for premedication and induction of anesthesia in patients with prolonged QT interval or those at high risk. Fentanyl also does not affect repolarization time at clinical doses, does not prolong QT and QTc distance at 2 μg/kg dose, and reduces QT prolongation when used before laryngoscopy and endotracheal intubation.[8] In this case report, induction of anesthesia was achieved using a combination of midazolam and fentanyl. To facilitate intubation, atracurium was administered as nondepolarizing neuromuscular blocking agent have

minor autonomic effects and is not associated with QT and QTc prolongation. Lidocaine was also administered before intubation to blunt the sympathetic response due to laryngoscopy.

The management of one-lung ventilation (OLV) must address intraoperative hypoxemia which is commonly recognized as a complication due to ventilation/perfusion mismatch. A frequent cause of hypoxemia is the dislocation of the device used for lung separation. Traditionally, solutions for the prevention and treatment of hypoxemia were the use of high fractions of inspired oxygen (FiO_2) and large tidal volumes but no positive end-expiratory pressure (PEEP) nor recruitment maneuvers (RM). It is reasonable to adopt the lowest possible FiO2, both before and during OLV to reduce resorption atelectasis in the ventilated lung and during reexpansion to reduce the oxidative stress in the nonventilated one. A consensus on the lowest safe limit of peripheral oxygen saturation to keep during OLV has not been reached, though most clinicians try to maintain it above or equal to 90%.

Protective ventilatory strategies are associated with reduced pulmonary and systemic inflammation, improved gas exchange, and fewer postoperative pulmonary complications. Regardless, the optimal tidal volume (TV) to adopt is unclear. While 5–6 mL/kg seems reasonable, some authors suggest 4–5 mL/kg, although not yet supported by evidence. The use of low TVs often leads to hypercapnia, which seems to exert protective effects against ventilator-induced lung injury. A $PaCO_2$ of 60–70 mmHg reached during OLV was not only well-tolerated, but linked to reduced post-thoracotomy lung and systemic inflammation. It appears reasonable to allow a certain degree of hypercapnia during OLV, except in patients with pulmonary or intracranial hypertension or major arrhythmias.

OLV seems to occur below the closing capacity of the ventilated lung. Strategies to maintain a certain end-expiratory volume are to be preferred. PEEP titration based on dynamic compliance of the respiratory system (with an average result of 10±2 cmH_2O) was shown to improve intraoperative oxygenation when compared with a fixed value of 5 cmH_2O. Studies comparing volume-controlled (VCV) with pressure-controlled ventilation (PCV) during OLV have led to equivocal

results in terms of oxygenation. Some authors support the use of PCV when high airway pressures are reached.[9] In this case report, a left side DLT was used to isolate the lung. The position of DLT was confirmed by auscultation, but the confirmation of the DLT position using flexible fiberoptic laryngoscopy was not performed because there was no change of position during procedure. Measurement of end tidal CO_2, peripheral oxygen saturation, and frequent blood sampling for blood gas analysis to monitor ventilation during procedure were performed to facilitate immediate the detection of conditions such as hypoxemia, hypercapnia, and hypocapnia.

CONCLUSION

CSD procedure often is considered as a final option to overcome refractory ventricular tachycardia, after pharmacological treatment with antiarrhythmic agents and catheter-based ablation have failed. Because patients whose undergoing CSD procedure are often in unfavorable conditions and may have complex comorbidities, preoperative preparation should be checked thoroughly and the anesthesiologist must obtain informed consent from the family regarding the risks of anesthesia including sudden cardiac death, and postoperative monitoring in intensive care unit.

This case report clarifies the significance of maintaining optimal hemodynamic, maintaining normovolemia, afterload reduction, adequate postoperative analgesia, and avoidance of cardiac depressant agents for successful perioperative anesthetic management for CSD procedure in patient with DCM. Anesthesiologists must determine the hemodynamic targets to be achieved before anesthetizing patients with dilated cardiomyopathy, so that several things must be appointed including the patient's clinical status and the degree of cardiac function, preparation of monitoring devices, anesthetic agents, and resources required to activate the ACLS protocol in the case of ventricular tachycardia and/or ventricular fibrillation perioperatively, which improve patient outcomes.

REFERENCES

1. Li M, Huang H. Anesthetic Management of Patients with Dilated Cardiomyopathy Undergoing Noncardiac Surgery.

Medicina (B Aires). 2023 Sep 1;59(9):1567.

2. Elsokkari I, Sapp JL. Electrical storm: Prognosis and management. Prog Cardiovasc Dis. 2021 May 1;66:70–9.
3. König S, Schröter T, Borger MA, Bertagnolli L, Nedios S, Darma A, et al. Outcomes following cardiac sympathetic denervation in patients with structural heart disease and refractory ventricular arrhythmia. EP Europace. 2022 Nov 22;24(11):1800–8.
4. Damasceno C, Delgado M, Pinheiro P, Quadros I. Anesthetic management in bilateral video-assisted thoracoscopic sympathectomy for refractory ventricular arrhythmias: A case report. Saudi J Anaesth. 2021 Apr 1;15(2):207.
5. Yadav N, Verma B, Aggarwal P, . R, Chiluka JDK. Anaesthetic Management of a Patient with Dilated Cardiomyopathy and Acute Decompensated Heart Failure. Journal of Clinical and Diagnostic Research. 2022 Jun 1;16(6):UD04–5.
6. Elliott IA, DeJesus M, Dobaria V, Vaseghi M, Ajijola OA, Shivkumar K, et al. Minimally Invasive Bilateral Stellate Ganglionectomy for Refractory Ventricular Tachycardia. JACC Clin Electrophysiol. 2021 Apr 1;7(4):533–5.
7. Pinsky MR, Cecconi M, Chew MS, De Backer D, Douglas I, Edwards M, et al. Effective hemodynamic monitoring. Crit Care. 2022 Dec 1;26(1).
8. Dede Ş, Demir ZA, Balcı E. Anesthesia induction regimens may affect QT interval in cardiac surgery patients: A randomized-controlled trial. Turkish Journal of

Thoracic and Cardiovascular Surgery. 2022;30(3):354.

9. Sun TT, Chen KX, Tao Y, Zhang GW, Zeng L, Lin M, et al. Effect of flow-optimized pressure control ventilation-volume guaranteed (PCV-VG) on postoperative pulmonary complications: a consort study. J Cardiothorac Surg. 2024 Dec 1;19(1):425.