European Review for Medical and Pharmacological Sciences 2017; 21: 627-634

Anesthetic management of patients with dilated cardiomyopathy for noncardiac surgery

C.-Q. CHEN[1], X. WANG[2], J. ZHANG[1], S.-M. ZHU[1]

[1]Department of Anesthesiology, the First Affiliated Hospital, School of Medicine, Zhejiang University, Hangzhou, People's Republic of China
[2]School of Medicine, Zhejiang University, Hang Zhou, People's Republic of China

Chao-qin Chen and *Xin Wang* contributed equally

Abstract. – Anesthetic management of patients with dilated cardiomyopathy (DCM) is a challenge to the anesthesiologist, due to poor left systolic function, ventricular enlargement, risk of malignant arrhythmias and sudden cardiac death. Therefore, preoperative assessment and appropriate anesthetic management are important in patients with DCM. This review describes the preoperative evaluation and anesthesia considerations of patients with DCM undergoing non-cardiac surgery. Patient pathophysiology and clinical status, such as ventricular function, degree of myocardial fibrosis, resting heart rate and high-sensitivity C-reactive protein can affect survival rates. Advanced monitoring devices, such as transesophageal echocardiography and cardiac resynchronization therapy can be used to assess ventricular function and myocardial fibrosis. Thoracic epidural blockade can improve ventricular function. In summary, the optimal anesthetic management of patients with dilated cardiomyopathy requires good preoperative assessment, close perioperative monitoring, suitable anesthetic, optimization fluid management, and stable hemodynamic status.

Key Words:
Dilated cardiomyopathy, Non-cardiac surgery, Systolic dysfunction, Anesthetic management.

Introduction

Dilated cardiomyopathy (DCM) is characterized by left ventricular or biventricular enlargement and impairment of systolic function. Anesthetic management of patients with DCM undergoing non-cardiac surgery is challenging and is associated with high mortality[1]. Unfortunately, there is a paucity of literature to guide anesthetic management for these patients. To aid anesthesiologists who care for these high-risk patients, anesthetic management is briefly reviewed.

The Features of Dilated Cardiomyopathy

DCM, a primary myocardial disease, is characterized by left or biventricular dilation, and systolic dysfunction, with or without congestive heart failure. DCM occurs in approximately 13/100000-84/100000 people and more frequently in males. The cause of DCM is unknown, although it may be associated with myocarditis, neuromuscular disorders, familial disease, idiopathic causes and other possible diseases. Previously, it was thought that the largest proportion of DCM was idiopathic (66%)[2]. Increasing evidence has shown that DCM has a familial basis[3]. Over 30 genes have been confirmed to be related to DCM[4], and sudden cardiac death in DCM was found to be associated with the long arm of chromosome 10. Mutations in the gene encoding lamin A/C were related to cardiac transplantation in DCM patients[5]. DCM 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 arrhythmias, heart failure, mitral or tricuspid regurgitation and sudden death. Although the 5-year mortality rate has decreased significantly, it was still 35-70% in children[6], and sudden death from DCM accounted for 30% of all deaths[7].

Preoperative Assessment

The preoperative assessment is very important in patients with DCM undergoing non-cardiac surgery. Electrocardiography (ECG) can be used to detect arrhythmia and evaluate the risk of sudden cardiac death (SCD). Left bundle branch block and prolonged QRS duration (> 120 ms) were independent predictors of increased mortality and SCD in heart failure patients[8,9]. Reduction

Corresponding Author: Sheng-mei Zhu, MD; e-mail: shengmei_zhu1@163.com

of resting heart rate (HR, < 80 beats/min) can reduce the risk of life-threatening arrhythmia, slow down myocardial remodeling, improve EF and New York Heart Association (NYHA) classification[10,11]. The value of mean QT and the slope of QT end and RR intervals (QT-slope) independently predict major arrhythmic events[12]. Holter ECG is a more favorable tool to detect arrhythmia, especially non-continuous arrhythmia. A previous study[13] showed that the occurrence of major arrhythmic events was the same, whether or not there was non-sustained ventricular tachycardia (nsVT). Whether heart rate variability and turbulence can predict arrhythmic events remains controversial[14,15]. Preoperative echocardiography is necessary to determine ventricular function and to assess the degree of valvular dysfunction[16]. International guidelines considered that patients with LVEF ≤ 35% and NYHA class I were a IIb class recommendation for ICD implantation, while NYHA class II or III patients with LVEF ≤ 35% were a class I recommendation[17]. Serum levels of B-type natriuretic peptide (BNP) have shown a correlation with left ventricular end-diastolic pressure, left ventricular wall stress, fibrosis and systolic dysfunction[18,19]. N-terminal (NT) pro-BNP levels over 2247 pmol/L were reported to be associated with higher mortality rates[20], but the utility of measuring NT pro-BNP levels has been questioned. Serum high-sensitivity CRP (hsCRP) level is also an independent predictor of survival rates in patients with DCM[20]. Ishikawa et al[21] reported that survival rates were significantly lower in patients with hsCRP levels over 1 mg/L. Also, lower EF, lower serum sodium, lymphocytopenia and higher serum creatinine have been reported to be independent predictors of transplantation or death in patients with DCM[22]. However, a lower EF in patients with DCM might have a normal CO. For example, if a patient has 30% EF, end diastolic volume of 250 ml, heart rate of 90 beats/min, and 30% regurgitant fraction, CO will be of 6.75 L/min and forward CO will be 4.725 L/min. Forward CO of 4.725 L/min may be sufficient for a 50 kg patient. However, if a patient presented with small left ventricular volume with low EF, the patient would have reduction in CO and a poor outcome[23]. A previous study[24] suggested that there were no significant differences between the preoperative NYHA classification and the incidence of complications. Cardiac magnetic resonance, a noninvasive imagining technique, has been used to detect myocardial fibrosis and predict survival rate in DCM[25-30]. Myocardial fibrosis can be found in at least one-third of patients with DCM, and it was previously shown that the mid-wall enhancement could predict SCD in patients with DCM[31-34].

Review of the medication history of the patient is important[35]. Many patients have been administered angiotensin-converting enzyme inhibitors (ACEI), β-adrenergic blockers and cardiotonic drugs to reduce afterload, slow ventricular remodeling and improve CO. β-adrenergic blockers should be used on the day of surgery to prevent the rebound phenomenon. Despite controversy, we also recommend that ACEI will be continued until the day of surgery, even if there is the possibility of intraoperative hypotension[36]. The serum potassium level should be evaluated because of the use of diuretics such as spironolactone. Cardiac resynchronization therapy (CRT) can reduce the morbidity and mortality of heart failure patients[37,38]. Biventricular pacing can improve left ventricular (LV) systolic function and decrease LV size and mitral regurgitation[39-41]. Therefore, if a patient has CRT, the function needs to be evaluated preoperatively.

Anesthetic Management

The key hemodynamic features of patients with DCM are elevated filling pressures, myocardial contractile dysfunction, and a marked negative relation between stroke volume and afterload[42]. Therefore, the anesthetic principles for DCM include[35,43-45]:

- Mmaintenance of myocardial contractility, avoiding drugs which can decrease myocardial contractility, maintenance of normal diastolic blood pressure to ensure coronary perfusion, maintenance of preload and preventing fluid overload;
- Prevention of increased afterload (systemic vascular resistance), avoidance of arrhythmias (i.e. tachycardia), and prevention of thromboembolic events.

Fluid Management

In DCM patients, the intraoperative fluid management should be cautiously managed. Because of poor cardiac ejection, ventricular enlargement, and elevated filling pressures, the fluid overload in the perioperative period could potentially lead to heart failure and pulmonary edema[46]. However, the fluid restriction can reduce CO. The adequacy of fluid management can be judged by central venous pressure (CVP), hemodynamics,

urine output, and serum lactate. A pulmonary artery wedge pressure of 12-15 mmHg or a CVP of 8-12 mmHg is recommended in cardiac surgery patients[23]. However, these pressures can be affected by various factors[47]. The transesophageal echocardiography (TEE) may be a more accurate tool to assess ventricular filling[48], although it is prohibited in patients with esophageal lesions and coagulopathy. The anesthesiologist should consider the amount of fluids which have been administered preoperatively when they determine the amount of fluid to infuse. A previous study[49] showed that large volumes of crystalloid fluid might cause the pulmonary edema easier, it might reduce the tissue oxygen supply, and it might affect the wound healing. Therefore, the amount of crystalloid should be controlled, and blood or blood products can be infused if necessary. The furosemide can be administrated to prevent the volume overload.

Mechanical Ventilation Settings

The mechanical ventilation may reduce venous return and CO, especially in the case of insufficient capacity[50]. In contrast, large tidal volume (TV) can reduce cardiac filling[51]; therefore, an appropriate TV (6-8 ml/kg) can be applied. In patients with DCM requiring mechanical ventilation, the application of positive end-expiratory pressure can improve CO in patients with elevated filling pressures, but has adverse effects on CO in patients with low pulmonary capillary wedge pressure[52].

Anesthesia Options

When we select the mode of anesthesia, the key point is to avoid myocardial depression, maintain hemodynamic stability, as well as meeting the requirements of surgery. Epidural anesthesia (EA) can reduce afterload and help to maintain forward flow from the left ventricle[44]. Another advantage of EA is that it can provide effective postoperative analgesia. However, a large dose of local anesthesia may cause a reduction in SVR and impairment of myocardial function. Therefore, slow administration of low-dose of local anesthesia or slow titration have been recommended to avoid rapid and extensive sympathetic nerve block[53]. Echigoya et al[54] suggested that mepivacaine (2 ml/kg) continuous infusion can be used in patients with DCM. EA with fentanyl has been confirmed to reduce afterload, improve cardiac function, and is accompanied by slow sympathetic blockade[55]. Hashimoto et al[56] suggested that fentanyl (10 μg/kg) can be administered intrathecally. Okutomi et al[57] showed that LVEF changed minimally after EA with bupivacaine. Intermittent boluses of bupivacaine (0.0625%) with fentanyl (2 μg/ml) could provide adequate analgesia with stable hemodynamic status[58]. Previous studies[59,60] have shown that high thoracic epidural sympathetic blockade can decrease left ventricular end-diastolic dimension, improve LVEF and NYHA classification, and reduce re-hospitalization rate.

General anesthetics chosen should have minimal inhibition on cardiovascular function according to the status of patients, and the dose administered. Induction of general anesthesia should be administered with small doses, and increased gradually according to the response of the patients.

Intravenous Anesthetics

Etomidate is often advocated as an induction anesthetic in patients with cardiac dysfunction because it causes minimal impact on cardiovascular function. Etomidate (0.3 mg/kg) did not cause a change in mean arterial pressure in children with congenital heart disease[61]. Although arterial pressure is maintained during etomidate anesthesia, a previous study[62] showed that etomidate anesthesia could cause increased left ventricular afterload, and affect myocardial function in patients with impaired LV function. 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[63], 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[64]. Dexmedetomidine, an 2-adrenoceptor agonist, was selected as a sedative and anxiolytic drug because it has sympatholytic and cardio-protective effects, without respiratory depression[65]. Dexmedetomidine can increase LV pressure, and stroke volume[66]. However, dexmedetomidine can slow the HR, which may reduce CO, especially in patients where the CO mainly depends on HR.

Volatile Anesthetics

Though inhalational anesthesia has the advantages of being quickly adjustable, and easily changing hemodynamic parameters, it may alter ventricular function because of strong myocar-

dial depression and preload reduction[67]. Isoflurane has been shown to reduce SVR in NYHA class II and III patients with coronary artery disease[68,69]. However, another study[70] suggested that 1.1-1.5 MAC isoflurane reduced CO and mean arterial pressure, and did not have beneficial effects on LV afterload in the presence of LV dysfunction. Sevoflurane (8%) has been found to cause a second degree atrioventricular block[71]. Ibrahim[72] reported that the induction of anesthesia with 3% sevoflurane and a bolus (1 μg/kg) of remifentanil in a child with DCM caused severe cardiovascular collapse. Adequate anesthetic depth is important to prevent the overload of afterload. Inhalational anesthesia at a low concentration (0.5-1.0 MAC) with a small dose (2-3 μg/kg) of fentanyl may be safely used because it did not decrease myocardial contractility[73]. Rylova et al[74] reported on a successful case of xenon anesthesia use in a patient with DCM, but further research on xenon anesthesia is required.

Opioids

Opioids (fentanyl and sufentanil) have minimal side effects on cardiac function[75]. Adequate HR and preload are necessary to maintain CO in patients with DCM. Remifentanil can cause bradycardia, especially in patients with anesthesia, on pure oxygen inhalational and using β-adrenergic blockade[76]. Morphine may cause decreasing preload and/or systemic vascular resistance (SVR). Application of large doses of all opioids must be carefully monitored.

Intraoperative Monitoring and Vasoactive Drugs

Suitable monitoring is needed to maintain an appropriate hemodynamics state. Invasive arterial monitoring, central venous pressure monitoring, pulmonary artery pressure monitoring, TEE, pacemakers, defibrillators, and bispectral index can be used during surgery according to the state of the patient. If a patient has an acute circulatory failure during surgery, appropriate vasoactive agents and fluid should be administered. Norepinephrine, epinephrine, or phenylephrine can increase mean arterial pressure. Norepinephrine can increase coronary perfusion by increasing diastolic blood pressure, while cardiac toxicity can be induced if the infusion is prolonged. Phenylephrine can worsen right ventricular function in patients with pulmonary hypertension[77]. If inotropic agents are required, milrinone, dobutamine, dopamine and low dose epinephrine can be used. All of those agents can improve the stroke volume, can increase the HR, and can decrease the filing pressure, while at the same time they can increase the cardiac work and the oxygen consumption[78,79]. Due to myocardial degeneration and fibrosis, digoxin should be administrated in small doses for poor tolerance. β-adrenergic agonists are the optimal vasoactive agent for DCM. Several anesthesiologists recommend pump infusion of dopamine before induction of anesthesia, to shorten the time of hypotension after induction.

Postoperative Analgesia

Good postoperative pain management must maintain postoperative hemodynamic stability, avoid cardiac depression, and avoid increasing SVR and HR. Combined application of local anesthetics, nonsteroidal anti-inflammatory drugs and opioids are often used for postoperative analgesia[80]. Epidural analgesia may be the best method for effective analgesia, with few adverse events[81,82]. Previous studies[83-85] have demonstrated that postoperative thoracic epidural analgesia can improve heart function in patients with heart failure and reduce perioperative adverse cardiac events. Epidural with fentanyl may be able to provide rapid analgesia.

Conclusions

This review focused on the preoperative evaluation and anesthesia considerations of patients with DCM. The optimal anesthetic management must assess the patient's pathophysiology and clinical status, select an appropriate method of anesthesia based on the kind of surgery and the degree of cardiac function, strengthen perioperative monitoring, select suitable anesthetics, and maintain management of optimization fluid and stable hemodynamic status.

Acknowledgements

We would like to thank Yue-ying Zheng for her suggestions with the study.

Sources of Funding

This work was supported by the Department of Anesthesiology, the First Affiliated Hospital, School of Medicine, Zhejiang University, Hang Zhou, China.

Conflict of Interest

The Authors declare that there are no conflicts of interest.

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