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Anesthetic management of a patient with severe dilated cardiomyopathy and an automatic implantable cardioverter-defibrillator (AICD) during total gastrectomy

Mamoru Takeuchi, Tatsuo Iwasaki, Tomoyo Oue, Naoyuki Taga, Yuichiro Toda, Katsunori Oe, and Kiyoshi Morita

Department of Anesthesiology and Resuscitology, Okayama University Graduate School of Medicine and Dentistry, 2-5-1 Shikata-cho, Okayama 700-8558, Japan

Abstract
We report a case of severe dilated cardiomyopathy with an automatic implantable cardioverter-defibrillator (ICD) undergoing total gastrectomy. During the operation, the defibrillation function of the ICD was suspended and its pacing function was used solely in VOO mode. Electrodes of an external defibrillator were attached on the chest wall, and a pulmonary arterial (PA) catheter with a ventricle pacing port was inserted into the pulmonary artery. Proper perioperative management, including measures that the patient underwent the surgery uneventfully and could attain a rapid and successful discharge from the intensive care unit.

Key words Dilated cardiomyopathy · Implantable cardioverter-defibrillator · Total gastrectomy

Introduction

Although many reports have addressed anesthetic managements for surgery for implantable cardioverter-defibrillator (ICD) implantation, only a few reports have specifically addressed these of patients who previously had implantation of ICD. We describe anesthetic and postoperative managements of a patient with severe dilated cardiomyopathy, who had previously received an automatic ICD (AICD), undergoing total gastrectomy.

Case report

A 63-year-old man, 171 cm tall and weighing 64 kg, was diagnosed of dilated cardiomyopathy (DCM) in 1986. He was first noticed to develop attacks of ventricular tachycardia (VT) accompanied by syncope in 1989. Antiarrhythmic drugs did not prevent VT; therefore, an AICD was implanted in February 1990. However, amiodarone was administered orally because VT had occurred frequently since 2000. Progressive anemia was noted during outpatient treatment, which was associated with worsening heart failure. The patient was admitted to our hospital in January 2001. A hemorrhagic advanced cancer was discovered in the fundus of his stomach. Total gastrectomy was scheduled in July 2001.

Upon physical examination, the patient's arterial blood pressure was 85/50 mmHg, his pulse rate was 70 beats·min^{-1} (ventricle pacing rhythm), and his New York Heart Association (NYHA) classification was level III. Preoperative chest radiography showed a cardiothoracic ratio of 59% and pulmonary congestion in both lungs. His hemoglobin level was 12.9 g·dl^{-1}, suggesting improvement of anemia by blood transfusion. Biochemical tests showed a maximum normal creatinine level (1.1 mg·dl^{-1}) but no other abnormalities. Coagulation tests and arterial blood gas analysis were normal. The electrocardiogram showed ventricle-pacing rhythm. Echocardiography demonstrated marked dilatation of the left atrium and left ventricle; mitral, tricuspid, and pulmonary valve regurgitation; advanced asynergy; and dysfunction. The ejection fraction and fractional shortening were reduced to 25% and 12%, respectively. The diameter of the inferior vena cava was 8 mm at inspiration and 17 mm at expiration. Neither pericardial fluid stagnation nor cardiac thrombus was observed.

Induction and maintenance of anesthesia

No premedication was applied. Anesthesia was induced with 750 μg of fentanyl and 10 mg of vecuronium. It was maintained with oxygen, nitrous oxide, and fentanyl. Electrodes of an external defibrillator that allow endermic pacing were attached. A catheter with a ventricle pacing port was inserted in the pulmonary artery (PA)

Address correspondence to: M. Takeuchi
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to facilitate arrhythmic control. During the operation, the defibrillation function of the ICD was suspended, whereas its pacing function was used exclusively in VOO mode. Dopamine at 3–5 $\mu g \cdot kg^{-1} \cdot min^{-1}$, dobutamine at 3–5 $\mu g \cdot kg^{-1} \cdot min^{-1}$, and milrinone at 0.2–0.4 $\mu g \cdot kg^{-1} \cdot min^{-1}$ were applied. The cardiac index was 1.71 $\cdot min^{-1} \cdot m^{-2}$ at the beginning of the operation, and was maintained at 1.7–2.41 $\cdot min^{-1} \cdot m^{-2}$ using colloidal solution and inotropic drugs. Systolic pressure was maintained at 80 mmHg, which was close to the value before the operation. Although human atrial natriuretic peptide was applied continuously for oliguria, no increase in urinary output was observed; urinary output during the operation was 35 ml. During the operation, VT attacks occurred twice; the first stopped naturally, whereas the second was terminated by burst pacing. During the entire operation, we used 1500 μg of fentanyl, 2300 ml of infusion, and 600 ml of packed cells. The operation time was 2 h 20 min, and the anesthesia time was 3 h 40 min.

Postoperative course

After admission to the intensive care unit (ICU), VT occurred, triggering the ICD on every occasion, followed by awakening. To prevent further VT, the patient was sedated with 600 μg of fentanyl and 0.5 $\mu g \cdot kg^{-1} \cdot min^{-1}$ of midazolam, and continuous infusion of 0.2–0.3 $\mu g \cdot kg^{-1} \cdot min^{-1}$ of nifekalant was started. This resulted in the disappearance of the VT attacks. Although application of various diuretics, such as continuous infusion (10–20 $mg \cdot h^{-1}$) of furosemide and intravenous injection of mannitol, did not increase the urinary output initially, these medications became effective about 15 h after ICU admission. The quantity of the inotropic drugs was decreased on the next day. Sedation was stopped, and the patient was discharged from the ICU after extubation.

Discussion

Key issues in the perioperative management of a patient whose condition is complicated by DCM include the control of heart failure and arrhythmia. This case, in addition to serious DCM, addresses issues of VT attacks and after ICD implantation. Although many reports address anesthetization in the ICD implantation operation [1–3], only a few reports have specifically addressed anesthetization of patients after implantation [4]. A patient carrying an implanted ICD is burdened by the hazard of ICD malfunction during other operations. Malfunction of an ICD during an operation is engendered mainly by electromagnetic interference [3], mostly resulting from the use of electrotomes.

Fundamental measures to mitigate this issue include avoiding the use of electrotomes during surgery or, alternatively, suspension of the ICD. In our case, the defibrillation function of the ICD was suspended during the operation; only its pacing function was used in VOO mode. Equipment that generates a pulse current, such as a peripheral nerve stimulator or an evoked potential monitor, aside from the electrotome, must be used carefully because it may cause ICD malfunction [5]. Moreover, electrolyte anomaly, especially that from potassium, should be avoided because it may cause the action of an ICD to fail [5]. In addition, operation posture, positive pressure ventilation, shivering, and dimensional change of the heart caused by changes in preload or afterload may cause malpositioning of the transvenous lead and consequently render the ICD inoperable [5]. Therefore, for prompt treatment of any VT or ventricular fibrillation, it is important for the external defibrillator to be available on standby. As a preparation for VT attacks, electrodes of an external defibrillator that allows endermic pacing were attached, and a PA catheter with a ventricle pacing port was inserted. In fact, VT attacks occurred twice during this operation; the first stopped naturally, whereas the second was terminated by burst pacing.

It is important for the control of heart failure to maintain cardiac contractility and circulating blood volume, and to prevent increase of afterload. In our case, narcotics known to cause only a small amount of circulatory depression were mainly used as anesthetics, and nitrous oxide was used exclusively for inhalation anesthesia. Furthermore, the blood pressure was suppressed to prevent any increases in cardiac work. Full monitoring is required to maintain an appropriate circulating blood volume. In this regard, the transesophageal echocardiogram is effective [6], but it was unavailable for an operation for stomach cancer in our patient. Therefore a PA catheter was inserted to monitor central venous pressure and pulmonary capillary wedge pressure. Adequate depth of anesthesia must be provided first to prevent any increase in afterload. The use of a vasodilator is also recommended [7]. Milrinone, a phosphodiesterase III inhibitor, was administered in this case. The cardiac index increased from 1.71 to 2.41 $\cdot min^{-1} \cdot m^{-2}$ after commencement of milrinone infusion.

In our case, amiodarone was administered orally before the start of the operation, which resulted in a total control of arrhythmia. The long half-life period of amiodarone suggests that the frequent VT attacks in the postoperative period were caused not by cessation of amiodarone but by exogenous catecholamine such as dopamine and endogenous catecholamine generated by operation-related stress. Therefore, midazolam and fentanyl were applied for sedation, and nifekalant was administered continuously until the level of catecholamine

was decreased. These medications were suspended after the decrease of exogenous catecholamine. Arrhythmia was well controlled thereafter.

Nifekalant, a pure K channel blocker, is administered intravenously. It prolongs action potential duration, prolongs the refractory period, and also prevents reentry. It also has a positive inotropic action and can be applied for deterioration of cardiac function [8]. In our case, no change of cardiac function was observed after commencement of nifekalant. However, this medication can potentially cause marked QT prolongation and torsades de pointes, especially during bradycardia [5]. For those reasons, it should be applied with care.

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