The Anesthetic Management for Emergency Operation on a Patient with Dilated Cardiomyopathy

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Dilated cardiomyopathy (DCM) is defined as a myocardial disease of unknown etiology, demonstrating the ventricular dilatation and broad hypokinetic wall motion. Although several cases of the anesthetic management for hypertrophic obstructive cardiomyopathy (HOCM), the one type of the idiopathic cardiomyopathy, have been reported, few anesthetic management for patients with DCM have been reported in Japan.

We describe here the anesthetic management for patients with DCM.

Case Report

A 25-year-old, 95 kg male was admitted to our hospital, complaining of right lower abdominal pain. A tenderness with muscular defense at the right lower abdomen was recognized. A diagnosis of acute appendicitis was made. He was showing dyspnea, sweating and palpitation. Body temperature, blood pressure and heart rate were 38°C, 110/74 mmHg and 130 bpm, respectively. His respiratory rate was 30/min under orthopnea. An apical systolic murmur of grade II/VI was present. Lower extremities were edematous. He had been treated with diuretics and digitalis for DCM for 2 years.

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Laboratory data showed leukocytosis, anemia and liver dysfunction. Echocardiography demonstrated marked dilatation of the left ventricle with poor wall motion. Diastolic left ventricular posterior wall thickness (LVPWT) was 11 mm and the ejection fraction (EF) was 19%. The chest X ray film showed marked cardiomegaly and a cardiothoracic ratio was 69%. The electrocardiogram showed sinus tachycardia and ST-depression in leads II, III, aVF, V_5 and V_6.

First, he was treated with antibiotics, but because of deterioration of abdominal symptoms, an emergency operation on appendicitis was scheduled. The patient did not receive premedication. Under local anesthesia, the right radial artery was cannulated to monitor arterial pressure and analyze blood gases. Anesthesia was induced with intravenous fentanyl 1.0 mg and diazepam 10 mg. Eighty mg of lidocain was intravenously administered to prevent ventricular dysrythmia. Muscle relaxation for tracheal intubation and operation was provided with 8 mg of pancuronium.

Anesthesia was maintained with N_2O 3 $l \cdot min^{-1}$, O_2 3 $l \cdot min^{-1}$, and fentanyl and diazepam as required. In order to monitor central venous pressure, a catheter was inserted into the superior caval vein via the right internal jugular vein. During the whole course of anesthesia, neither hypotension nor dysrhythmia was observed. After

the operation, he was admitted to the intensive care unit. On the first postoperative day the trachea was extubated, and on the third postoperative day he uneventfully returned to the ward. On the seventh postoperative day, when skin sutures were removed the operative wound dehisced. An emergency operation for the dehisced wound was scheduled. Induction of anesthesia with intravenous ketamine of 100 mg was followed by severe hypotension, but 15 min later, blood pressure almost returned spontaneously to a pre-induction level. Anesthesia was maintained with N_2O 3 $l \cdot min^{-1}$, O_2 3 $l \cdot min^{-1}$ and supplementary doses of fentanyl. Muscle relaxation for tracheal intubation and operation was provided with 8 mg of pancuronium. The anesthesia course thereafter was uneventful. The patient was admitted to the intensive care unit. On the first postoperative day the trachea was extubated and on the third postoperative day he returned to the ward. the postoperative course was satisfactory and the wound healed. However, two months after the second operation, he died of congestive heart failure.

Discussion

Idiopathic cardiomyopathy is classified into three categories: dilated cardiomyopathy (DCM), hypertrophic cardiomyopathy (HCM) and restrictive cardiomyopathy (RCM)[1].

DCM is characterized by diminished myocardial contractility and dilatation of the left or right ventricle, or both the ventricles[1]. An autosomal recessive inheritance[2], disorder of myocardial metabolism, poisoning and myocarditis are suspected as etiologies. Some of DCM are due probably to myocarditis, because the microscopic examination of the myocardial biopsy specimen shows inflammatory cell infiltration and gallium accumulates to the myocardium in the gallium scintigraphy[3]. The main cause of death in DCM is heart failure or sudden death. DCM has a poor prognosis with a survival rate of 54% at the age of 5 and 36% at the age of 10[2]. On the electrocardiogram ventricular premature beats and atrial fibrillation are common[4], and anesthesia may increase the rate of appearance of dysrythmias or aggravate dysrhythmias. There is no satisfactory treatment except for cardiac transplantation because of unknown etiology. The mainstays of the treatment for symptoms are bedrest, digitalis and diuretics.

Inotropic agents such as catecholamines, vasodilators, β-blockers and immunodepressants are also used. A current topic of the treatment for DCM is low-dose β-blockade therapy[5,6]. In heart failure, subsensitivity to cathecholamines occurs as a consequence of a reduced β-adrenergic receptor density, and high levels of catecholamine may produce direct cardiotoxic effect. Beta-adrenergic receptors are down-regulated in patients with congestive heart failure, and myocardial catecholamines are depleted even though their serum levels are high[5,6]. Gradual dose titration of β-adrenergic blocking agents may provide time for up-regulation of β-adrenergic receptors to take place. This would allow restoration of cathecholamine responsiveness and improved myocardial function[7]. But it is difficult to use β-adrenergic blockers during anesthesia for patients with DCM, because it is important to avoid cardiovascular depression in the anesthetic management for DCM. Local, spinal or epidural anesthesia may be selected according to the sites of operation. In spinal anesthesia or epidural anesthesia, it goes without saying that hypotension should be avoided. In general anesthesia, volatile anesthetics which produce myocardial depression must be avoided. Therefore, the narcotics is recommended because of its slight effect on myocardial contractility[8]. In this patient, the first anesthesia was uneventfully conducted, but the second anesthesia in which ketamine was used for the induction induced severe hypotension.

It has been considered that ketamine is ideal for the induction of anesthesia in the presence of congestive heart failure[9]. Ketamine increases heart rate, blood pressure, cardiac output and pulmonary artery pressure via stimulation of the sympathetic nervous system, diminution of fre-

quency response of baroreceptors[10], and inhibition of the intraneuronal reuptake of norepinephrine[11]. However, ketamine has a direct cardiovascular depressive effect[12], therefore, may produce undesirable cardiovascular depression when catecholamine depletion occurs[13]. Ketamine may also reduce cardiac output if exessive catecholamine exposure exists by activation of the sympathetic nervous system, because of its biphasic effects on myocardium, i.e., cardiovascular stimulation and direct cardiovascular depression with high doses[12]. The sympathetic nervous system may be chronically hyperactivated in DCM[14]. There is a report concerning ketamine that the blood pressure was maximally decreased between the fourth and seventh minutes and returned to a normal level by the tenth minutes under halothane anesthesia[15]. These support that severe hypotension which lasted for about 15 min in this patient was induced by ketamine. Therefore, ketamine must be carefully used when the sympathetic nervous system is hyperactivated.

We reported the twice-conducted anesthetic management for the same patient with DCM. In the anesthetic management for the patient with DCM, the use of ketamine should be restrained, and narcotics with minimal effects on myocardial contractility should be used.

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