Anesthetic management of patients with arrhythmogenic right ventricular dysplasia/cardiomyopathy: Two case reports

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Citation

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Abstract

We used typical anesthetic techniques for anesthetic management of patients with arrhythmogenic right ventricular dysplasia/cardiomyopathy (ARVD/C) undergoing abdominal and gynecological surgery. The patients, a 54-year-old man and a 22-year-old woman, had ventricular tachycardia (VT), and the symptoms and physical findings were controlled by medication. Anesthetic induction was performed using propofol and fentanyl, and tracheal intubation was facilitated by the use of vecuronium. Anesthetic maintenance was performed using sevoflurane and nitrous oxide combined with epidural anesthesia. Methoxamine was administered to control mild hypotension; the patient's hemodynamic parameters were stable, and VT was absent. The use of intravenous antiarrhythmic agents for treatment or the use of a defibrillator was not required. We think that 1) the symptoms and physical findings were well controlled and 2) the avoidance of tachycardia by removal of noxious stimuli and limited administration of positive chronotropic agents are important for anesthetic management of patients with known ARVD/C.

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INTRODUCTION

Although arrhythmogenic right ventricular dysplasia/cardiomyopathy (ARVD/C) is known to be one of the main causes of sudden unexpected perioperative death, few studies have reported the anesthetic management of patients with known ARVD/C because of its rare prevalence [1–3]. We performed anesthetic management of 2 patients by using typical anesthetic techniques to prevent lethal arrhythmia during surgery.

CASE DESCRIPTION

The first patient was a 54-year-old man (height, 161 cm and weight, 56 kg) who was under medication for ventricular tachycardia (VT) for 5 years. He had a benign pancreatic tumor and epigastric pain and was scheduled for pylorus-preserving pancreaticoduodenectomy. VT recurred after admission, and more than 10 combinations of 11 types of antiarrhythmic agents were tested to determine the effective drugs. During the course of treatment, the cardiologist made the following findings: a combination of atenolol, flecainide, and bepridil hydrochloride was better at controlling arrhythmia; VT did not cause severe hypotension; and intravenous administration of lidocaine and dipyridamole was effective for treating VT. Medication control caused a decrease in arrhythmias. Premature ventricular contraction (PVC) reduced from 14944 times/day (tpd) to 129 tpd, couplets reduced from 64 tpd to 1 tpd, triplets reduced from 3 tpd to 0 tpd, and VT reduced from 12 tpd to 0 tpd. Transesophageal echocardiography revealed segmental wall motion abnormality (SWMA) at the basolateral wall in the right ventricle (RV), and RV angiography revealed diffuse RV SWMA, especially reduced inferior wall motion. A cardiac muscle biopsy revealed fat infiltration in the right ventricle. In the electrophysiological examination, we could reproducibly induce VT similar to natural VT, and the reentry circuit indicated by electroanatomical mapping could not be repaired by catheter ablation.

The second patient was a 22-year-old woman (height, 158 cm and weight, 64 kg) who had had no symptoms and

physical findings until PVC was detected 6 years ago. She experienced chest tightness 2 years ago and had a syncope-like episode 4 months ago; she was admitted to a hospital. She was scheduled for laparoscopic ovariectomy of an ovarian tumor that was opportunistically diagnosed by screening. Electrocardiography revealed negative T wave in the leads V1–V3 and biphasic T wave in the leads II, III, aVL, and aVF. Holter electrocardiography revealed PVC of 4830 tpd, couplets of 139 tpd, triplets of 8 tpd, and VT of 2 tpd; of the 2 instances of VT, one was characterized by 14 consecutive ventricular beats and the other by less than 14. Transthoracic echocardiography revealed mild hypokinesis in the left ventricular apex, aneurysm in the right ventricular apex, reduced right ventricular wall motion, trabecular derangement, and trivial tricuspid regurgitation. A cardiac muscle biopsy revealed fat infiltration in the right ventricle. Electrophysiologic examination revealed delayed conduction, where VT could not be induced, on the septal side and periphery of the right ventricular apex as well as in the septum of the left ventricle. Propranolol was selected as the medication control after testing 5 combinations of 2 antiarrhythmic agents.

In addition to the daily dose of antiarrhythmic agents, patient 1 was intramuscularly administered 20 mg of famotidine and patient 2 was intramuscularly administered 2.5 mg of midazolam and 20 mg of famotidine as premedication on the morning of surgery. Epidural catheters were placed into the intervertebral space at Th10/11 and L1/2. Anesthetic induction was performed using 80 mg of propofol and 200 μg of fentanyl. Vecuronium (6 mg) was used to facilitate tracheal intubation and oxygen mixed with 50–66% nitrous oxide was used for mechanical ventilation. Anesthesia was maintained using 1.0–2.5% sevoflurane combined with epidural anesthesia. Mepivacaine (1%) and lidocaine (1.5%) was administered into the epidural space of patients 1 and 2, respectively, during surgery. Continuous infusion of lidocaine at a dose of 0.5 mg/kg/h was performed to prevent arrhythmias in patient 1. Neostigmine (2 or 2.5 mg) and atropine (1.0 mg) were slowly administered to reverse the effect of the muscle relaxant. Emergence from anesthesia was smooth, and extubation was performed without problems. Hemodynamic parameters were stable, and only simple intervention using methoxamine (α_1 adrenergic receptor agonist) was required against mild hypotension; VT did not occur throughout the surgery. Patient 1 had an episode of VT 3 days after surgery and was treated by lidocaine and dipyridamole.

DISCUSSION

ARVD/C is an inherited disease characterized by structural and functional abnormalities that involve mostly the right ventricle and can cause life-threatening ventricular arrhythmias in apparently healthy young people [1, 2, 4]. Although no data regarding the anesthesia protocol are available, we think that attention should be paid to the following aspects. Firstly, elective surgery should be conducted only after arrhythmia and other symptoms are controlled and the patient's condition is stable. The use of an implantable cardioverter-defibrillator may also be suitable [2]. In our patients, medication control was attempted well before surgery, and the patients took antiarrhythmic agents until the morning of surgery. These factors may have contributed to the prevention of lethal arrhythmia during surgery. In addition to medication, knowledge of the symptoms, physical findings, and effective treatment for VT are desirable. We were prepared to administer intravenous antiarrhythmic agents, such as lidocaine and dipyridamole, to patient 1 because of their effectiveness against VT and to use the defibrillator if the need arose. Secondly, since tachycardia can cause VT, it is thought that noxious stimuli such as anxiolysis before surgery, pain, and administration of positive chronotropic drugs should be avoided as much as possible. For avoiding the induction of VT, we provided sufficient analgesia by combining balanced general anesthesia with epidural anesthesia, selected methoxamine as a pressor, and paid careful attention to slow administration of neostigmine with atropine for reversing the effect of the muscle relaxant. If the patients had heart failure due to severely reduced contraction, the dose or kind of anesthetics should be changed to preserve heart function.

We performed anesthetic management of 2 patients with ARVD/C, in whom typical general anesthesia combined with epidural anesthesia did not cause VT during surgery.

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