Accelerated idioventricular rhythm associated with desflurane anesthesia
— A case report —

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Accelerated idioventricular rhythm is defined as a ventricular rhythm of 60-100 beats per minute or a ventricular tachycardia that does not exceed 120 beats per minute. Although, it rarely converts to a fatal arrhythmia like ventricular fibrillation, it needs to be differentiated from AIVR, which is from another origin. AIVR may occur due to ischemic heart disease (ST elevated myocardial infarction), cardiomyopathy, rheumatic fever and digitalis intoxication. We report here on a case of AIVR that was related to desflurane administration. (Korean J Anesthesiol 2009; 56: 571~3)

Key Words: Accelerated idioventricular rhythm, Arrhythmia, Desflurane, Sevoflurane.

The term of accelerated idioventricular rhythm (AIVR) or slow ventricular tachycardia describes an ectopic ventricular rhythm with 3 or more consecutive ventricular premature beats with a rate faster than the normal ventricular intrinsic escape rate of 30 to 40 beats per minute, but slower than ventricular tachycardia [1]. Although AIVR has been reported in patients during administration of inhaled anesthetic, desflurane, it is converted to normal sinus rhythm after stopping administration of desflurane [2]. We described here a patient who experienced AIVR during the induction of anesthesia for the total knee replacement arthroplasty (TKRA) in the right knee that persisted after change of anesthetic from desflurane to sevoflurane.

CASE REPORT

A 63-year-old woman (weight 77 kg, height 165 cm) was scheduled for elective TKRA owing to osteoarthritis in her right knee. At her preoperative visit, she reported no history of cardiovascular disease and routine preoperative laboratory test (blood cell count, hemoglobin, coagulation test, serum glucose, nitrogen, creatine, electrolyte, bilirubin, aspartic acid transaminase (AST), alanine transaminase (ALT), creatine kinase, lactate dehydrogenase, cholinesterase, and urine analysis) were within normal range.

The patient had previously undergone two operations for excision of varicose veins in her lower legs; both operations ended without complications. Her vital signs at the admission were stable (blood pressure 134−120/89−83 mmHg, pulse rate 54 bpm, body temperature 36.9°C). Her preoperative ECG showed first degree AV block (Fig. 1). Echocardiogram showed normal systolic function (EF 63%) and no structural or functional abnormalities. A thallium scan showed that perfusion of the myocardium was normal. She received no premedication. On arrival in the operating room, her blood pressure was 140/80 mmHg and pulse rate was 75 bpm. Thiopental sodium 300 mg was administered to induce unconsciousness and rocuronium 40 mg was injected. Mask ventilation was performed with desflurane and nitrous oxide (N2O). As the end tidal concentration of desflurane approached to 5.2 vol%, an abnormal EKG wave was observed at a rate of 60−80 bpm for 10 sec accompanied by a wide QRS complex (Fig. 2) at 5-10-second intervals. Despite administration of lidocaine 100 mg, the ar-
rhythmia persisted (blood pressure 100−90/65−48 mmHg, pulse rate 60−80 bpm). Then, the patient was intubated with 7.0 sized endotracheal tube under the direct laryngoscopy. Arterial blood gas analysis showed that her acid-base status and electrolyte levels were normal.

There were no specific, identifiable organic problems, so we inferred that the arrhythmia was due to sympathetic activation accompanying the increased end tidal concentration of desflurane. After stopping the inhalation of desflurane and nitrous oxide, the dysrhythmia disappeared spontaneously as the concentration of desflurane was reduced to less than 2.0 vol%. When inhalation of desflurane and nitrous oxide was restarted however, an idioventricular rhythm characterized by wide QRS with alternating three different waves occurred when end tidal concentration of desflurane had increased to 3.5 vol%, suggesting that desflurane was the cause of the arrhythmia. Although there were no alterations in vital sign (blood pressure 120−100/65−50 mmHg, pulse rate 75−50 bpm), we stopped the administration of desflurane and administered sevoflurane.

There were no significant alterations to vital signs (blood pressure 110−100/60−52 mmHg, pulse rate 70 bpm), so the operation was continued under sevoflurane anesthesia. AIVR is frequently associated with reperfusion of the myocardium after myocardial infarction [3], so we assessed the patient’s postoperative troponin I (TnI), troponin T (TnT), creatine kinase, and myoglobin; all were within normal ranges. The patient got recovered normal sinus rhythm at the recovery room after about 20 minutes from the end of operation. A postoperative EKG also did not show any abnormalities. The patient was discharged on postoperative day 10 without any further problems.

**DISCUSSION**

Ventricular arrhythmias during anesthesia are caused by previous cardiac disease, electrolyte disturbances or in relation to drugs given intraoperatively [4]. AIVR may occur when an atrial pacemaker slows down or when the automaticity of a pacemaker in the ventricle accelerates [5]. The electrophysiologic characteristics of AIVR show that it is not a re-entrant arrhythmia but is due to the abnormal activation of automaticity caused by increased phase 4 depolarization in ventricular muscle fibers [6]. Generally, AIVR is of short duration, disappearing gradually after 30 beats. Also, conversion to fatal arrhythmia such as ventricular arrhythmia is rare [2].

AIVR occurs in 25% of patients with ST-elevated myocardial infarction (STEMI), especially during fibrinolytic therapy at the time of reperfusion [7]. AIVR can also occur following cardiac operations; in patients with cardiomyopathy, rheumatic fever, or digitalis intoxication; and in patients with no evidence of cardiac disease [8].

AIVR has also been observed during the induction of anesthesia with desflurane, disappearing after replacement of desflurane with propofol [6]. Besides that, AIVR has also been reported to develop under spinal anesthesia recovering to a normal sinus rhythm after administration of atropine [2]. In another report about AIVR, bupivacaine can induce AIVR without preceding central nervous system toxicity [9]. In this case, the authors said that bupivacaine might injure myocardium and AIVR could occur as a result of direct myocardial injury [9].

We originally thought that AIVR in our patient was likely caused by the sympathomimetic effect of desflurane on the heart because the arrhythmia disappeared when desflurane inhalation was stopped. However AIVR persisted after the change to sevoflurane, so other causes are likely.

Although desflurane has low solubility, making the induction and emergence of anesthesia rapid, a rapid increase in its concentration can induce symptoms related to increased sympathetic activity, including hypertension and tachycardia [10].
This is due to desflurane activation of the sympathetic nervous system and increases in intramyocardial norepinephrine and epinephrine [11-13]. Epinephrine increase the rate of phase 4 depolarization in automatic cell, which can induce arrhythmia such as AIVR. Desflurane has been reported to prolong the QT interval, which is thought to be cause of AIVR [14]. The QTc interval is an index of the depolarization and repolarization of myocardium. Although a direct causal relationship between AIVR and QTc interval has not been shown, they are likely related because both are related to depolarization of the left ventricle.

In contrast to desflurane, sevoflurane does not induce arrhythmias by stimulating sympathetic nervous system. However sevoflurane has been shown to sensitize the heart and reduce the threshold of epinephrine concentration at which arrhythmia are induced [15]. Therefore it could be inferred that arrhythmia might occur if sevoflurane is administered when the concentration of catecholamine is increased. Therefore an arrhythmia may occur if sevoflurane is administered when the concentration of catecholamine is increased.

We suspected the occurrence of coronary vasospasm and reperfusion, so we measured the level of cardiac enzyme and performed a follow-up EKG, but there was no evidence of ischemic heart disease. We therefore propose that AIVR occurred in our patient owing to the rapid increase in desflurane concentration; AIVR persisted after the change of desflurane to sevoflurane because sevoflurane maintained heart sensitization owing to the increased levels of intramyocardial catecholamine.

AIVR rarely converts to fatal ventricular arrhythmia, so treatment of AIVR usually involves observing vital signs and performing EKGs until the patient recovers a normal sinus rhythm. Patient who show persistent hypotension and bradycardia should be treated with an alpha agonist or atropine [5].

In conclusion, AIVR may be induced by activation of sympathetic nervous system that is caused by a rapid increase in desflurane concentration and may be maintained when desflurane anesthetic is replaced by another inhalation agent such as sevoflurane.

REFERENCES