症 例

Ventricular tachycardia occurred in a hypothermic patient during emergence from general anesthesia

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Summary

A 76-year-old man underwent trans-urethral resection of bladder tumor and laparoscopic total nephroureterectomy of the left renal pelvic and bladder tumor under general anesthesia combined with lumbar epidural anesthesia. Premature ventricular contractions and subsequent ventricular tachycardia occurred at emergence from anesthesia, when his rectal temperature was 33.5℃. Following cardiac massage and lidocaine administration, normal sinus rhythm was obtained. The arrhythmias were suspected to be mainly due to an increase of ventricular alpha- and beta-adrenoreceptor activation induced by perioperative hypothermia and mild hypercapnia.

Key words; hypothermia, ventricular tachycardia, torsade de pointes, general anesthesia, epidural anesthesia

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Introduction

The perioperative maintenance of normothermia is associated with a reduced incidence of morbid cardiac events and ventricular tachycardia[1], and perioperative cardiovascular morbidity continues to be an important clinical problem for most patients being considered for major noncardiac surgery[2,3]. Advanced age, peripheral vascular arteriosclerosis, scheduled abdominal surgical procedure, open leg position, and general and epidural combined anesthesia have been reported to be risks for the perioperative dysrhythmias[1]. We report ventricular tachycardia possibly induced by hypothermia in an elderly patient undergoing urological surgery under general and epidural anesthesia.

Case report

A 76-year-old man (154.3cm, 44.2kg) was scheduled for trans-urethral resection of bladder tumor and laparoscopic total nephroureterectomy of the left renal pelvic and bladder tumor. The preoperative condition of the patient was American Society Anesthesiologists (ASA) II, New York Heart Association Classification 1, and we had not observed any medical history about dyspnea or chest pain on effort. The patient had not received beta-blockade or any other medication preoperatively. Electrocardiography and chest X-rays showed no abnormal findings. Hematologic laboratory values were also within the normal range. In the ward, his heart rate and blood pressure

were 76 beats per minutes and 134/75 (94) mmHg, respectively, and his body temperature measured in the axillary was 36.4℃, and the core temperature in the operating room measured in the eardrum was 36.3℃. An epidural catheter was placed at the lumbar 3-4 intervertebral space before induction of general anesthesia, and 1% lidocaine 5ml and 100μg of fentanyl were administered, followed by continuous infusion of 1% lidocaine at 5-7ml/hour via the catheter. General anesthesia was induced with 5mg droperidol, 2μg/kg fentanyl, 1.5mg/kg propofol, and 0.2mg/kg vecuronium, and maintained with 1.5-2% sevoflurane and 50% nitrous oxide in oxygen under controlled ventilation to maintain the $PaCO_2$ at between 35 and 40mmHg. The patient was monitored with II-lead electrocardiograph, left radial arterial blood pressure, pulse-oxymetry, and end-tidal capnogram (Anesthesia intelligent monitoring system™, Helsinki, Finland). All fluid lines were warmed during the operation, and a heat-moisture exchange device and a circulating water mattress (T-pump, Gaymar industries, Inc., NY, USA) were set at 38.5℃.

First, trans-urethral resection of bladder tumor was performed for 45 minutes in the open leg position, and second, total laparoscopic nephrectomy followed by open ureterectomy was performed for the next 3 hours and 37 minutes in the right lateral jack-knife position. Rectal temperature decreased to 33.5℃ after trans-urethral resection of bladder tumor, and was kept at 33.2-33.3℃ until completion of surgery. After surgery, forced-air warming was started, and the patient was ventilated with 100% oxygen. The oxygen saturation (SpO_2) on the right foot showed 97%, and that on the left foot 70%. The heart rate and blood pressure were 63 beats per minute and 113/62mmHg, respectively. After confirming spontaneous respiration, atropine 1.0mg and neostigmine 2.0mg were administered. Approximately 10 minutes later, 5-6 premature ventricular contractions per minute were recognized, and then, electrocardiograph was changed to ventricular tachycardia (**Fig. 1**). After cardiac massage (120 times/min) and intravenous

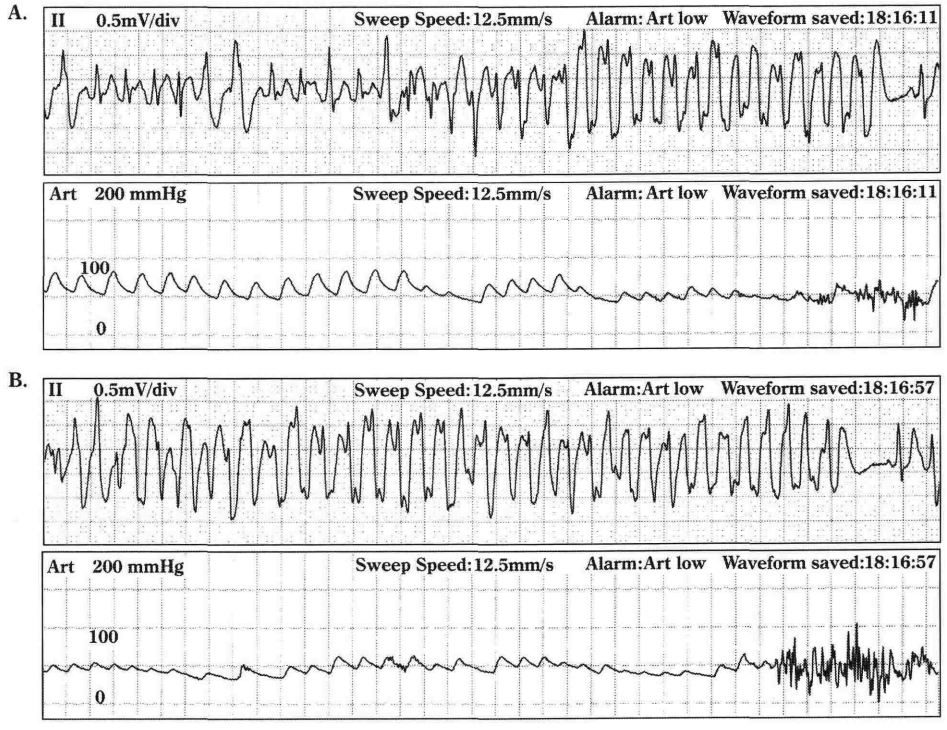

Figure 1 Ventricular Tachycardia and radial artery blood pressure
Electrocardiogram (II-lead) and left radial artery blood pressure during the episode of ventricular tachycardia. Figure 1-A and 1-B are chronological.

administration of 2mg/kg of 2% lidocaine, ventricular tachycardia disappeared and normal sinus rhythm was obtained within 1 minute. Arterial blood gas analysis at this point indicated pH: 7.301, $PaCO_2$: 7.0kPa (53mmHg), PaO_2: 62.6kPa (471mmHg), and HCO_3^-: 26mmol/l. Other electrolyte and cell blood count data were Na^+: 140mmol/l, K^+: 3.5mmol/l, Ca^{2+}: 1.16mmol/l, Ht 31%, and Hb 11g/dl.

After 15 minutes of the episode, although the rectal temperature was still low (33.9℃), the temperatures of eardrum and the axillary were 35.6℃ and 36.4℃, respectively. Then, the patient was extubated. No arrhythmia or any ischemic changes were observed in the 12 leads of the electrocardiogram, and the heart rate and blood pressure were 72 beats per minute and 127/71mmHg, respectively. SpO_2 showed recovery to 100% in both right and left feet. Intraoperatively administered total fluid was 2500ml, urine output was 850ml, and bleeding was 57ml.

Discussions

We presented a case of ventricular tachycardia that occurred in a hypothermic patient during emergence from anesthesia. We considered that the main cause of ventricular tachycardia was hypothermia under general and epidural combined anesthesia. Hypothermia is an independent predictor of morbid cardiac events from a multivariate analysis, indicating a 55% reduction of risk when normothermia is maintained[1]. The patient was at high risk of postoperative hypothermia and subsequent arrhythmia, because of advanced age, peripheral vascular arteriosclerosis, scheduled abdominal surgical procedure, open leg position, and general and epidural combined anesthesia[1,4]. The incidence of hypothermia is more frequent in patients under regional block combined with general anesthesia than the patients with general anesthesia alone[4]. The patient's hypothermic state started just after the induction of general anesthesia and continued to the end of surgery (surgical procedure: about 5 hours). We could not ignore the possibility that the temperature of the fluids used during resection of the tumor was not appropriately warmed up to 37–40℃. Abrupt increase of eardrum temperature might be attributed to the sensor being affected by forced-air warming.

We should have used forced-air warming from the beginning of surgery. Forced-air warming is more effective for maintaining body temperature than circulating water mattresses, airway heating and humidification, or intravenous fluid warming[5], and decreases the incidence of shivering[6] and adrenergic response[7]. Ventricular tachycardia occurred just after the cessation of general anesthesia and administration of neostigmine and atropine. The patient should have been kept under general anesthesia in controlled ventilation without reversing the effect of muscle relaxant until the core temperature recovered to at least 36.5–37.0°C. In a hypothermic patient, anesthetics may protect patients from the incidence of ventricular tachycardia by attenuating alpha- and beta-adrenergic activation, such as: 1) potentiation of norepinephrine response[7], 2) alpha-mediated ventricular ectopy due to the increased delayed after depolarization, which triggers increased activity in Purkinje fibers[8], and 3) beta-adrenoreceptor activation by norepinephrine[9].

The causes of ventricular tachycardia are considered to be abnormalities of the myocardial ion channel, which are aggravated by sympathetic nerve activation, psychotropic drugs, antibiotics, anticancer drugs, electrolyte abnormalities (such as hypokalemia, hypocalcemia, or hypomagnesemia), myocardial potassium channel blocking, anti-hyperlipidemia drug, cerebral hemorrhage, ischemic coronary disease, mitral valve disease, hypothermia, and hypothyroidism[10]. In the present case, the K^+ level was 3.5mmol/l and the Ca^{2+} level was 1.16mmol/l, and hypokalemia and hypocalcemia might have contributed to a cause of ventricular tachycardia. Although we did not measure the Mg^{2+} level, we could not ignore the possibility of hypomagnesemia as a contributor of this arrhythmia.

We should discuss other possible factors that promote the occurrence of ventricular tachycardia. First, in the arterial blood gas analysis after recovery from ventricular tachycardia, $PaCO_2$ was 53mmHg, and hypercapnia might have promoted ventricular tachy-

cardia. Second, QT prolongation and serious arrhythmia have been reported in patients receiving droperidol[11,12], and we cannot neglect the possibility that droperidol might be one of the causes of the induction of ventricular tachycardia in the present case, although arrhythmia occurred after several hours following administration of droperidol.

In the present case, SpO_2 on the right foot was 97%, and on the left foot was 70% when surgery was completed. The reason why the data on the left foot were lower than that of on the right foot is not clear. However, the constriction of the peripheral vasculature of the left foot due to hypothermia and the right lateral jack-knife position might have caused the dissociation of data between the right and left feet.

In conclusion, we experienced a case that showed ventricular tachycardia during emergence from general anesthesia. We considered that arrhythmias were mainly induced by ventricular alpha- and beta-adrenoreceptor activation under perioperative hypothermia and mild hypercapnia after reversal of muscular relaxant. It might be recommended to maintain general anesthesia until recovery to normothermia, especially in elderly patients.

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