Coronary Artery Spasm Immediately Following Extubation of the Trachea

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It is now generally accepted that coronary artery spasm plays an important role in the genesis of myocardial ischemia[1,2]. Perioperative coronary artery spasm appears to be observed most frequently after myocardial revascularization in patients undergoing coronary artery bypass surgery[3,4,5]. In this report, we present a case of coronary artery spasm in noncardiac surgery, which occurred just after extubation of the trachea.

Case History

A 75-yr-old man (height, 156 cm; weight, 47 kg) with a rest pain at the lower extremities and intermittent claudication (50m) was diagnosed as arteriosclerosis obliterans and was scheduled to undergo an elective right axillo-bifemoral bypass. His past medical history consisted of essential hypertension diagnosed 40 years before and an attack of hemiplegia probably due to a cerebral hemorrhage 25 years earlier: he had already been relieved from the hemiplegia on admission. Medical therapy consisted of salt restriction (8 $g \cdot day^{-1}$) and nifedipine (15 mg p.o.$\cdot day^{-1}$) for the control of hypertension. He had never experienced any cardiac symptoms (angina, palpitation, dyspnea etc). He had a history of cigarette smoking (1 package/day, for 55 yr). His family history was unremarkable. Physical examination on admission revealed no abnormal findings with regard to his cardiopulmonary function. Preoperative hemogram and blood chemistries were normal. Pulmonary function tests were within normal limits. A chest X-ray revealed a cardiothoracic ratio of 48%. A preoperative 12-lead ECG at rest showed left ventricular hypertrophy ($SV_1+RV_5=44$ mV) without an apparent finding of myocardial ischemia[6]. A treadmill exercise test could not be performed because of his claudication.

On the morning of surgery, he received nifedipine (5 mg) as usual, and he was premedicated with nitrazepam 2 mg orally 2 hours before induction of anesthesia. Intraoperatively, he was monitored using a radial artery catheter, a central venous pressure (CVP) catheter, ECG (modified lead

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II^7 [II^m] & lead V5), rectal and skin temperature probe, pulse oxymeter and capnometer. Preinduction vital signs were a respiratory rate of 12 min, a heart rate of 65 beats·min^{-1} and blood pressure of 165/85 mmHg. The ECG monitor showed regular sinus rhythm with a sporadic appearance of premature supraventricular beats (1–2 beats·min^{-1}), though he was asymptomatic. After 5 min preoxygenation, anesthesia was induced with thiamylal 150 mg, fentanyl 0.15 mg, and diazepam 2 mg. The trachea was intubated after administration of pancuronium 4 mg. Anesthesia was maintained with fentanyl 0.4 mg, diazepam 4 mg and 50% N_2O/0.5–1.2% enflurance in oxygen. Since he had long suffered from hypertension and had a history of cerebral vascular accident, during the surgery, in order to ensure cerebral blood flow and also the bypass flow, the systolic blood pressure was maintained between 120 and 150 mmHg with the aid of dopamine infusion (3–6 μg·kg^{-1}·min^{-1}), monitoring CVP and maintaining the adequate preload. During the surgery, the hemodynamic variables were stable except for a sporadic appearance of premature supraventicular beats. The operation time was 225 min. The total blood loss was 1,200g: the hemoglobin (Hb) at the end of the surgery was 9.7 g·dl^{-1} (hematocrit [Ht]=31%). Blood transfusion was not performed. Before extubation of the trachea, rectal and skin (lt. thumb) temperatures were 35.9°C and 31.5°C, respectively and the following prerequisites for extubation were confirmed: 1) recovery from neuromuscular blockade by a nerve stimulator; 2) adequacy of ventilation and oxygenation by a measurement of mintue ventilation (5.5 l·min^{-1}; a respiratory rate of 16 min) and blood gas analysis (pH 7.38, Pa_{CO_2} 38.1 mmHg, Pa_{O_2} 228 mmHg [F_{IO_2}=0.5], BE −1.8); and 3) awakening of the patient (confirmed just before the extubation). For about 8 min before extubation of the trachea, the lungs were ventilated with 100% oxygen without tracheal suctioning and during this time, his hemodynamic variables were stable: heart rate; 90 beats ·min^{-1}, blood pressure; 135/65 mmHg and ECG; regular sinus rhythm without ST deviation (lead II^m & V5). After suctioning of the pharynx, the trachea was extubated with the following method, which has been generally recommended[7,8]: it was accomplished by inflating his lungs with about 30 cmH_2O pressure, holding the pressure constant while the cuff is rapidly deflated, and then rapidly pulling the tube out. Just after the extubation, ST segment elevation (0.4 mV) occurred in the lead II^m (fig. 1B-b), immediately followed by a decrease in blood pressure (60–80/30–50 mmHg) and multiple occurrence of both supraventricular and ventricular (multifocal, bigeminy, short run) premature beats. The patient complained of neither chest pain nor discomfort, but he mentioned some mild lower limb pain. Since the occurrence of coronary artery spasm was strongly suspected from its characteristic onset and the ECG change, the following therapy was conducted under inhalation of 100% oxygen: 1) continuous infusions of nitroglycerin (0.5–0.8 μg·kg^{-1}·min^{-1}) and diltiazem 0.5–0.8 μg·kg^{-1}·min^{-1}); 2) a continuous dopamine infusion (10–16 μg·kg^{-1}·min^{-1}) in an attempt to increase blood pressure; and 3) one shot administration of lidocaine (40 mg) followed by a continuous lidocaine infusion (40–80 mg·hr^{-1}) against the ventricular arrhythmia. The results of arterial blood analysis one minute after the ECG change were as follows: pH 7.42, Pa_{CO_2} 34.3 mmHg, Pa_{O_2} 381.7 mmHg, BE −1.1, Hb 9.8 g·dl^{-1}, Ht 32%, Na 135.2 mEq·l^{-1}, K 3.5 mEq·l^{-1}, Cl 110 mEq·l^{-1}, Ca 1.02

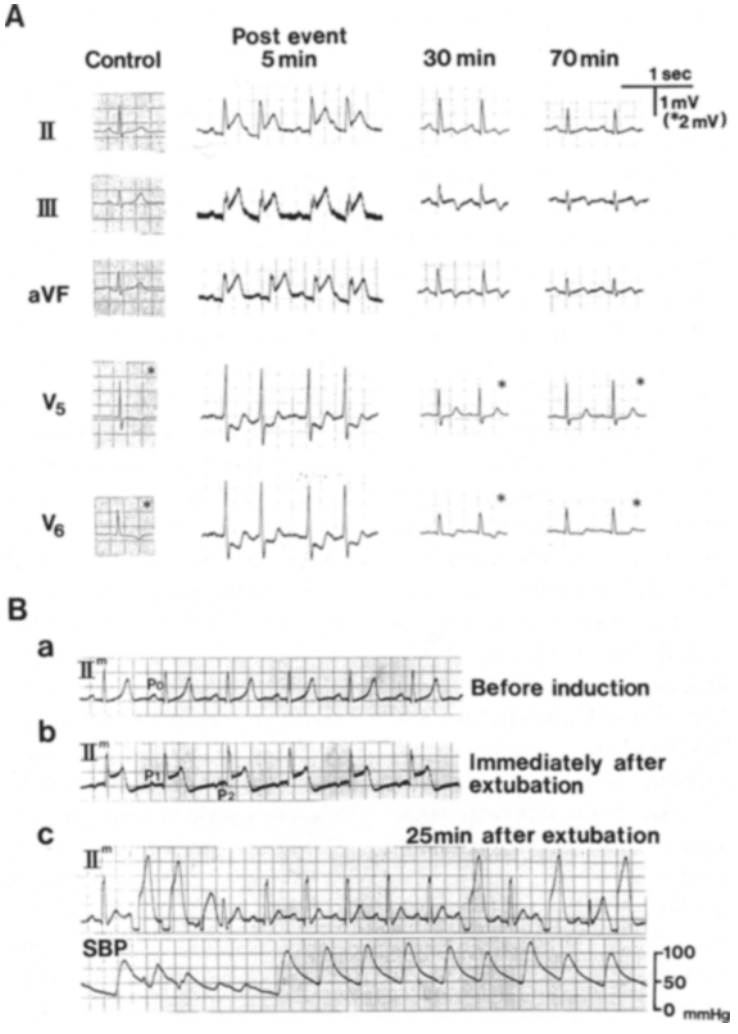

Fig. 1. A: Time course of ECG changes after extubation of the trachea in leads II, III, aVF, V5. V6 (control ECG was recorded on admission). B-a: The ECG in the modified lead II (II^m) before induction of the anesthesia. B-b: The ECG (lead II^m) recording immediately after extubation of the trachea, showing a significant ST-segment elevation and changes in P wave (P_1, P_2 vs P_0). B-c: The ECG recording 25 min after extubation of the trachea (lead II^m). Although the ST-segment already almost returned to the baseline, the arrhythmias (ventricular premature beats) were still observed. Abbreviations: SBP=systemic blood pressure (radial artery), II^m=modified lead II (from right shoulder to cardiac apex[7]).

mEq·l^{-1}, lactate 16.7 mg·dl^{-1} (before the induction; 12.5 mg·dl^{-1}, and blood sugar 210 mg·dl^{-1}. Five mintues after the ECG change, a 12 leads ECG was recorded and showed significant ST segment elevation in leads II, III, and aVF and depression in leads I, aVL, and V4–6. Since the CVP route was used as the infusion route of the above drugs, CVP was not monitored during the hemodynamic collapse. About ten minutes after starting the therapy, the

ST segment began to return towards the baseline, and almost completely returned to the baseline 20 min after starting the therapy. However, the arrhythmias persisted for about 30 min even after the recovery of ST segment and were resistant to lidocaine (fig. 1B-c). The duration of the hypotension below 80 mmHg (systolic) was about 5 min. After the blood pressure increased, the dose of dopamine infusion could be gradually reduced (16→4 $\mu g \cdot kg^{-1} \cdot min^{-1}$) and morphine was administered intravenously in an attempt to relieve his lower limb pain (total dose 5 mg: intermittently administered observing the hemodynamic change). After evaluating the CVP (5 mmHg), blood transfusion was conducted in order to ensure the oxygen delivery to his vital organs (Hb; 9.8→11.5 $g \cdot dl^{-1}$). After the hemodynamic state became stable, he was moved to the Intensive Care Unit (ICU) with continous infusions of the following drugs: nitroglycerin (0.5 $\mu g \cdot kg^{-1} \cdot min^{-1}$), diltiazem (0.8 $\mu g \cdot kg^{-1} \cdot min^{-1}$), lidocaine (60 $mg \cdot hr^{-1}$) and dopamine (4 $\mu g \cdot kg^{-1} \cdot min^{-1}$). In the ICU, his hemodynamic state was quite stable. An echocadiogram performed in the ICU revealed no abnormal findings: wall motion and ejection fraction of his heart were within normal limits. Under intensive observation, the above drugs were carefully weaned. CPK-MB from the blood sampled postoperatively (1 and 17 hours after the event) was negative. The bypass blood flow was ensured by a Doppler flowmeter. Next morning, he was discharged from the ICU. His postoperative course was uneventful. There was no difference between the ECG before the operation and that obtained 2 days later.

Discussion

Although a definitive diagnosis of coronary artery spasm requires the demonstration of focal spasms by coronary arteriography, the sudden ST segment changes observed in this patient strongly suggested the occurrence of coronary artery spasm due to the following reasons: (1) The ST-segment elevated in the leads II, III, aVF. It is known that the ST-segment elevation indicates transmural myocardial ischemia caused by total or subtotal occlusion of a major coronary artery, which is not induced by increased myocardial demand alone in patients without old myocardial infarction (in patients with old myocardial infarction, ST-segment elevation can be induced by stress in leads where abnormal Q waves are present and may not necessarily indicate myocardial ischemia)[1]. Furthermore, it was reported[9,10] that right coronary artery spasm is more frequent than spasm of the left main, anterior descending, and the circumflex coronary artery and is commonly accompanied by ST-segment elevation in leads II, III, aVF. (2) The ST-segment changes developed without any preceding significant changes in the determinants of myocardial demand or supply[1,3].

The precise mechanism(s) of coronary artery spasm has not yet been fully elucidated: several causes of coronary artery spasm have been reported, including direct mechanical irritation, drug-induced effects (ergonovine, methacholine, acetylcholine), neurogenic influences, and atheromatous disease[1–3]. During the perioperative period, myocardial revascularization, hyperventilation, injection of calcium salts, low temperature and activation of vasovagal reflexes have been reported as the causes of coronary artery spasm[3,4,11,12]. Although it appears to still be controversial whether or not the stimulation of parasympathetic nervous system can really evoke coronary artery spasm[1,2,13], we speculate that in the present patient, the procedure of extubation of the tra-

chea evoked the coronary artery spasm probably by stimulating the parasympathetic (vagal) nervous system. The reasons for this speculation are as follows: 1) since the sensory and motor innervation of the most part of the airway is provided by the vagus nerve[14], it is quite conceivable that the procedure of extubation of trachea mechanically stimulates the vagus nerve, causing the activation of vagal-vagal reflexes; 2) the application of the positive airway pressure during the removal of the endotracheal tube might also activate the parasympathetic nervous system by stimulating the mechanoreceptor of the lung and thoracic wall; and 3) the stimulating effect of fentanyl on parasympathetic nervous system might still remain on the extubation of trachea. Another possibility might be that the application of the positive airway pressure during the extubation lead to the development of coronary artery spasm via mechanical oppression or transformation of the heart (major coronary artery) besides the activation of parasympathetic nervous system.

It is uncertain whether the ST-segment depression observed in lateral precordial leads was the reciprocal one reflecting the ST-segment elevation in leads II, III, aVF, or merely reflected the mild myocardial ischemia of the lateral wall due to coronary artery spasm, because ST-segment depression can develop as a result of coronary artery spasm depending on the severity of ischemia. The arrhythmias, which persistently occurred after the ST-segment return to baseline, were presumably well-known reperfusion-related arrhythmias.

In the present case, intravenous administration of nitroglycerin and diltiazem were effective in relieving the ST-segment change as has been reported in the previous studies[3,5,9]. Although the early detection of coronary artery spasm and the following rapid aggressive treatment are of great importance, as a recent review noted[3], the best treatment is prevention but such prevention requires a high level of suspicion that a coronary artery spasm is likely to occur; such suspicion relies on careful preoperative evaluation and awareness of certain situations during which a coronary artery spasm is more likely to occur. Although the present case had never previously experienced any cardiac symptom, he had various major risk factors for coronary artery disease, including atherosclerosis, aging, smoking history, and hypertension[15]. Therefore, we should have been more careful to evaluate the patient preoperatively and perioperative administration of nitroglycerin and/or Ca^{2+} antagonist might have prevented the event.

In conclusion, we report the sudden onset of coronary artery spasm immediately following extubation of the trachea. As the cause of this event, the stimulation of parasympathetic nervous system during the removal of the endotracheal tube was strongly suspected. In order to both prevent and detect early the occurrence of coronary artery spasm, it is important for anesthesiologists to be aware of various situations during which a coronary artery spasm is likely to occur. The extubation of trachea is presumably one such situation. Therfore, we recommend that extreme care should be taken on extubation of the trachea in patients with known and suspected coronary heart disease: e.g. intensive ST-segment monitoring, consideration of preventive administration of nitroglycerin and/or Ca^{2+} channel blocker, and consideration of the extubation of trachea without application of the positive airway pressure etc.

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