# Recurrent Coronary Artery Spasm during a Non-cardiac Surgical Procedure

Takahiko OKUDA and Katsutoshi WAKITA

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Coronary artery spasm is often reported in the perioperative period[1-11]. Myocardial ischemia caused by coronary artery spasm as characterized by ST-segment elevation occurs suddenly and is not preceded by an increase in blood pressure or heart rate. This may frequently result in premature contractions, atrioventricular block, severe hypotension and even cardiac arrest. We report a case of coronary artery spasm occurred 4 times during a noncardiac surgical procedure.

## Case History

A 65-year-old man (weight 56 kg, height 160 cm) was scheduled for colonostomy for a sigmoid concer. The patient had no history of ischemic heart disease. No abnormal finding was noted in the recent chest X-ray, electrocardiograph and other laboratory data. Physical examination revealed also no special finding. The patient was premedicated with 3 mg midazolam and 0.5 mg atropine intramuscularly one hour before induction of anesthesia. In the operating room, a needle was inserted at the L1-L2 interspace and an epidural catheter was placed cephalad through the needle. Anesthesia was induced with 5 mg increments of midazolam. Following loss of consciousness, vecuronium of 8 mg was administered intravenously and the tracha was intubated uneventfully with only a slight change in blood pressure and heart rate. Anesthesia was maintained with a combination of epidural anesthesia and general anesthesia with isoflurane, nitrous oxide and oxygen. Ventilation was controlled to maintain the $Pa_{CO_2}$ between 35 and 40 mmHg. Epidural anesthesia was induced with 6 ml of 2% lidocaine, following a test dose of 2 ml of 1% lidocaine. Fifteen minutes after injection of the lidocaine, arterial blood pressure gradually decreased from 120/60 to 80/40 mmHg and the heart rate from 90 to 70 bpm. The hypotension was treated with a rapid intravenous fluid infusion and dopamine of 5 $\mu g \cdot kg^{-1} \cdot min^{-1}$. Arterial blood pressure gradually increased to 120/50 mmHg and was maintained between 130/80 and 120/50 mmHg. Two and a half hours after the start of the operation, ST segment elevation appeared suddenly in lead II. One minute later, QRS complex widening, multifocal VPC and a short run of ventricular premature contractions (VPC) appeared (fig.1) and arterial blood pressure then decreased to 60/40 mmHg. Since coronary spasm was suspected, 0.2 mg bolus of nitroglycerin was injected over 10 min and followed

*Department of Anesthesiology, Kinki University School of Medicine, Osaka, Japan*

*Address reprint requests to Dr. Okuda: Department of Anesthesiology, Kinki University School of Medicine, 377-2, Onohigashi, Osakasayama-shi, Osaka, 589 Japan*

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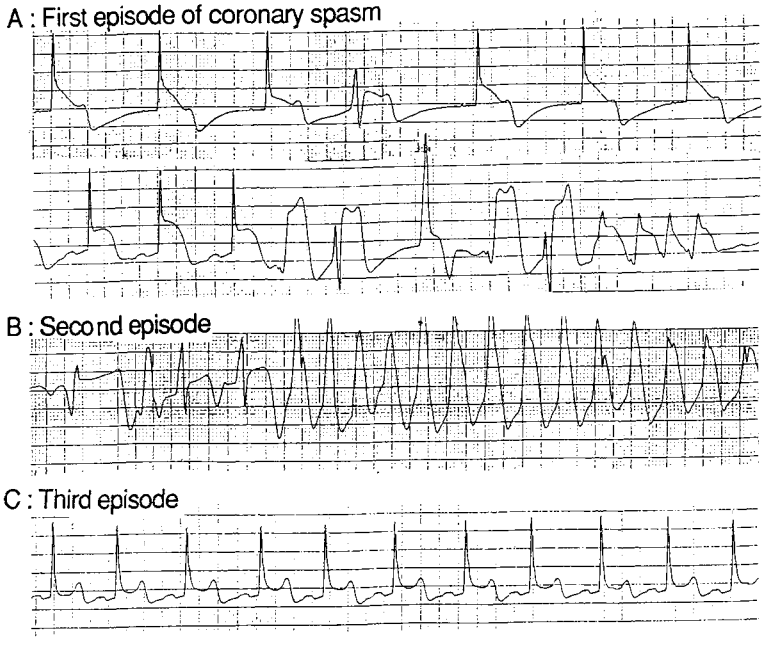


Fig. 1.

by a continuous infusion at a rate of 0.5 $\mu g \cdot kg^{-1} \cdot min^{-1}$. Lidocaine 1 $mg \cdot kg^{-1}$ was administered for the arrhythmias. Three minutes later, the ST segment elevation had returned to baseline and the electrocardiogram again showed sinus rhythm at a rate of 70 bpm. Then minutes after the first episode, a similar episode recurred again associated with VPC and VT. Diltiazem 10 mg and lidocaine 1 $mg \cdot kg^{-1}$ were immediately administered. Two minutes later, the ST segment elevation and ventricular dysrhythmias disappeared and the electrocardiogram again showed sinus rhythm. Other two episodes occurred in the later period of anesthesia. In the third and fourth episodes, slight ST elevation appeared merely and disappeared within one minute. An infusion of 0.5 $\mu g \cdot kg^{-1} \cdot min^{-1}$ of nitroglycerin and 3 $\mu g \cdot kg^{-1} \cdot min^{-1}$ of diltiazem were maintained throughtout the period of anesthesia. Postoperatively, the ECG remained within normal limits and serum enzyme levels (SGOT, LDH, CPK) were within normal range. No further episode of coronary spasm occurred in the perioperative period.

## Discussion

Sudden ST segment elevation occurred suspecting coronary spasm during general anesthesia in a patient without an antecedent increase in heart rate or systolic blood pressure. Although exact diagnosis requires arteriographic demonstration of reversal coronary obstruction, the hemodynamic and reversible electrocardiographic changes impressed us strongly for it.

It is suggested that coronary spasm is due to constriction of coronary vascular smooth muscle cells triggered by alterations of the autonomic nervous system, an increase in intracellular calcium ions, and vasoactive substances[12]. During general anesthesia, the most important risk factor is an autonomic nervous system instability. Stimulation of the sympathethic nervous system, e.g. light phase of anesthesia, hypoxia, and administration of cathecolamine or

atropine, can induce coronary spasm. Hyperventilation, which influences intracellular calcium ions, may play an important role in the production of coronary spasm[10,12]. In our case, $Pa_{CO_2}$ was maintained within normal range. Refractory excitation of the sympathetic nervous system responded to either hypotension or vagal stimulation played a important role in the production of coronary spasm. It has been shown that an epidural block affects the coronary spasm[1,3,7,8,11]. Coronary spasm under epidural or spinal anesthesia be classified into groups according to denervation of the cardiac sympathetic nervous system 1. One group is those with cardiac sympathetic nervous system blockade and the other is those without cardiac sympathetic nervous system blockade. In our case, cardiac sympathetic nerves were not blocked because the epidural catheter was inserted at the L1-L2 interspase. In group without cardiac sympathetic nervous system blockade, compensatory vasoconstriction is seen above the level of sympathetic blockade, presumably in an effort to offset the reduction in peripheral resistance caused by the block. This reflex sympathetic activity would involve the cardiac sympathetic nerves causing coronary vasoconstriction and consequent ischemia[1].

Several papers have reported cases of coronary artery spasm during abdominal surgery under epidural anesthesia supplemented with intravenous dopamin[1,3,8,11]. The administration of catecholamine, which stimulates alpha adrenergic receptors, to treat a decrease in arterial pressure due to sympathethic nervous system blockade and relative predominancy of the parasympathetic nervous system by the epidural block induces the development of coronary spasm. Most patients with variant angina are over the age of 40 and male. These patients have a high incidence of significant atherosclerotic coronary artery stenosis. This seems to indicate that coronary spasm is somehow related to atherosclerosis. Risk factors include hyperlipidemia, hypertension, smoking, and diabetes mellitus related atherosclerosis[12].

The consequences of coronary artery spasm induced ischemia require rapid treatment. Significant and prolonged vasospasm may lead to acute myocardial infarction and sudden circulatory arrest and death[4,5]. Administration of nitroglycerin and calcium antagonists may prevent coronary spasm. Continuous infusions of nitroglycerin at a rate of 0.5 to 1.0 $\mu g \cdot kg^{-1} \cdot min^{-1}$ have been recommended for its prophylaxis. In our case, recurrent attacks occurred in spite of a nitroglycerin infusion at a rate of 0.5 $\mu g \cdot kg^{-1} \cdot min^{-1}$. This dose might have been not sufficient to prevent coronary spasm. Although administration of nitroglycerin promptly relieves coronary spasm, this has not consistently resolved ST segment changes, hypotension, or arrhythmias. Calcium antagonists which dilate large coronary arteries are as effective as nitroglycerin and are useful to relieve coronary spasm[5,7–9].

In summary, a case of coronary spasm occurring non-cardiac surgery is described. Recurrent coronary spasm occurred during the anesthesia period. Nitroglycerin and a calcium antagonist were effective in management of the coronary spasm.

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## References

1. Krantz EM, Viljoen JF, Gilbert MS: Prinzmetal's variant angina during extradural anaesthesia. Br J Anaesth 54:945–949, 1980
2. Buffington CW, Ivey TD: Coronary artery spasm during general anesthesia. Anesthesiology 55:466–469, 1981
3. Koga M, Matsunaga M, Dan K, et al: Coronary artery spasm during up-

per abdominal surgery under epidural anesthesia. J Jap Soc Clin Anesth 3:44–51, 1983
4. Briard C, Coriat P, Commin P, et al: Coronary artery spasm during non-cardiac surgical procedure. Anaesthesia 38:467–470, 1983
5. Nussmeier NA, Slogoff S: Verapamil treatment of intraoperative coronary artery spasm. Anesthesiology 62:539–541, 1985
6. Yamanoue T, Mukaida K, Kawachi S, et al: Coronary spasms during non-cardiac surgery. Masui (Jpn J Anesthesiol) 35:1119–1125, 1986
7. Kano T, Ushijima K, Saito Y: Two cases of coronary artery spasm under epidural anesthesia. Masui (Jpn J Anesthesiol) 35:1896–1905, 1986
8. Nonaka A, Nakano S, Kumazawa T: Recurrent coronary artery spasm during anesthesia: A case report. J Jap Soc Clin Anesth 9:426–430, 1989
9. Tatara T, Kuroda T, Miyao H, et al: Coronary Spasm during laryngoplasty. Masui (Jpn J Anesthesiol) 39:99–105, 1990
10. Saito S, Dohi S, Naito H: Hyperventilation induced coronary artery spasm during anesthesia for neurosurgery. J Anesth 5:309–312, 1991
11. Mishima S, Kasai K, Yamamoto M, et al: Coronary artery spasm during abdominal surgery: Two episodes in the same patient. J Jap Soc Clin Anesth 11:333–339, 1991
12. Yasue H: Pathophysiology and treatment of coronary artery spasm. Chest (Supplement) 78:216–223, 1980