Isosorbide Dinitrate Attenuated Coronary Artery Spasm during General Anesthesia for Non-cardiac Surgery

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Isosorbide dinitrate (ISDN) is effective for congestive heart failure and unstable angina[1-3]. ISDN is also used during coronary arteriography[4] for the resolution of coronary artery spasm through a general inhibition of smooth muscle contraction. There have been several case reports on coronary artery spasm during general anesthesia successfully treated with intravenous administrations of nitroglycerin alone, or a combination of nitroglycerin and a calcium entry blocking drug[6-8]. However, the effect of ISDN on coronary artery spasm during general anesthesia has not been reported. This is a report of the occurrence of coronary artery spasm, suggested by marked ST-segment elevation and wide QRS complexes on electrocardiogram (ECG) during general anesthesia for non-cardiac surgery, which was dramatically attenuated by ISDN administration.

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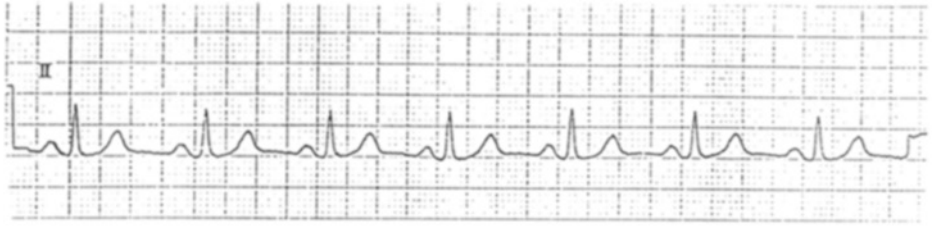
Case Report

A 67-yr-old man (67 kg) was admitted for sigmoidectomy. He has a history of vasospastic angina pectoris, gastric ulcer, pneumonia, chronic bronchitis, mild hypertension, and tobacco abuse (20 cigarettes daily for about 50 years). Vasospastic angina pectoris was diagnosed by anginal attacks early in the morning, but coronary arteriogram demonstrated 25% stenoses in segments 1 and 3 of the coronary arteries. The patient has been treated with oral diltiazem and ISDN, 60 mg twice daily for two years since the episode of angina pectoris. Physical examination and preoperative laboratory evaluation were unremarkable except a reduced forced expiratory volume in 1 second which was 2.14L (58% of predicted). ECG showed regular sinus rhythm with normal ST segment.

Two ISDN tapes were placed on the precordial region 6 hours before admission to the operating room and he was premedicated with pethidine 50 mg intramuscularly 30 min before arrival in the operating room. Arterial blood pressure and heart rate were 165/90 mmHg and 65 bpm, respectively, and ECG on the modified lead II showed



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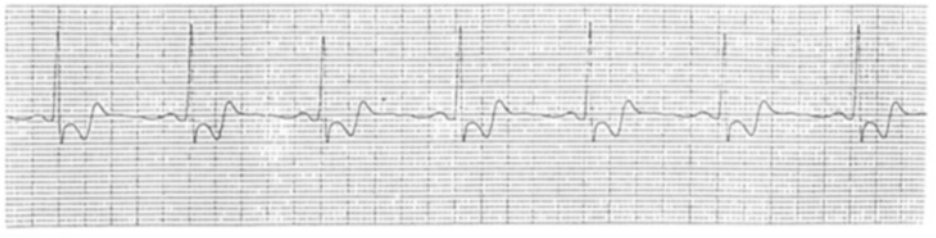

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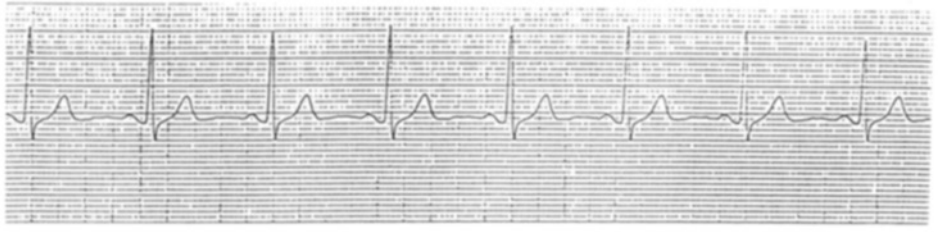

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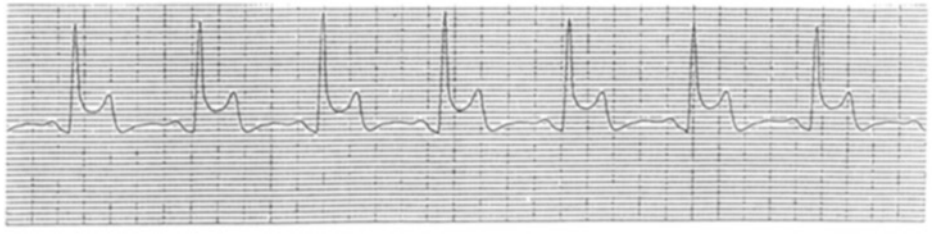

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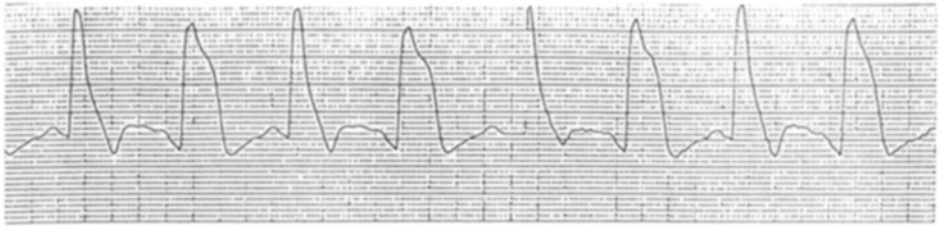

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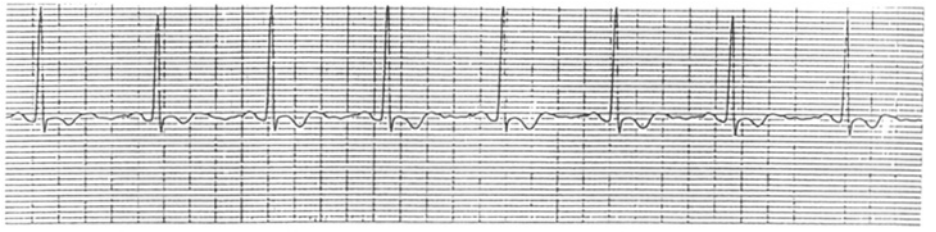

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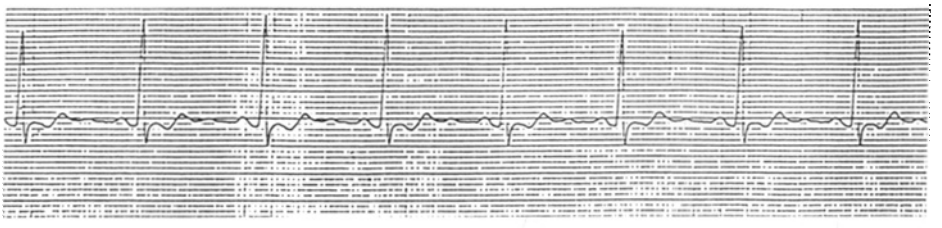

G

Fig. 1. ECG (lead II) changes during the episode
A Preanesthetic ECG in lead II.
B – ECG (lead II) of ST depression and T-wave inversion immediately after intubation.
C – ECG (lead II) of return of the ST segment to baseline.
D – ECG (lead II) of ST elevation 25 min after the start of surgery.
E – ECG (lead II) of progressive ST elevation with two alternating wide complexes.
F – ECG (lead II) of normal QRS complexes with inverted T waves.
G – ECG (lead II) of showing return of the inverted T waves almost to a normal pattern.

regular sinus rhythm with normal ST segment on admission to the operating room (fig. 1-A). A cannula was inserted at the right radial artery to measure arterial blood pressure, and an epidural catheter was inserted at the L1–L2 intervertebral space and advanced 5 cm cephalad. Anesthesia was induced with intravenous thiopental 4 $mg \cdot kg^{-1}$, diazepam 0.15 $mg \cdot kg^{-1}$, and the trachea was intubated with vecuronium 0.16 $mg \cdot kg^{-1}$. Immediately after intubation, blood pressure decreased to 110/60 mmHg, heart rate increased to 115 bpm, and the ECG showed progressive ST segment depression (fig. 1-B). Three min later intravenous administration of phenylephrine 0.05 mg and ISDN 1 mg resulted in immediate return of the ST segment to baseline (fig. 1-C), followed by stabilization of the hemodynamic variables.

Anesthesia was maintained with enflurane 0.5–1.0 vol% in nitrous oxide (3 $l \cdot min^{-1}$) and oxygen (3 $l \cdot min^{-1}$). Intermittent administration of 1% mepivacaine (2–3 ml) for epidural block was also performed. Ventilation was controlled to maintain Pa_{CO_2} between 35 and 40 torr and Pa_{O_2} over 100 torr. The operation was started after placing another ISDN tape on the chest. During lymph nodes resection 25 min after the start of surgery, systolic blood pressure decreased from 120 mmHg to 100mmHg with ST-segment elevation (fig. 1-D). We administered 0.5 mg of ISDN intravenously because

we thought the episode was caused by coronary artery spasm. But the ST-segment elevated further. Moreover, wide QRS complexes appeared 3 min after the ST-segment elevation (fig. 1-E). Intravenous ISDN was administered incrementally until the ECG showed normal QRS complexes. After administration of ISDN 3 mg, the ECG showed normal QRS complexes with inverted T wave within 6 min after the episode of the ST-segment elevation (fig. 1-F), and the inverted T wave returned to a normal pattern 1 min later (fig. 1-G). From that point until the end of the operation, ISDN was administered intravenously at a rate of 0.5 $\mu g \cdot kg^{-1} \cdot min^{-1}$. The operation and postoperative course went uneventful.

Discussion

Episodes of myocardial ischemia in this case were successfully treated with incremental intravenous administration of ISDN. Nitroglycerin (NTG) and calcium entry blocking drugs (e.g., nifedipine, verapamil) have been demonstrated to be effective in resolution of coronary artery spasm during general anesthesia[9–11]. The optimal dose and an adequate route of administration of NTG during coronary artery spasm are imperfectly elucidated, and systolic blood pressure sometimes decreases severely even with incremental intravenous administration of low doses. Although sublingual nifedipine is effective in resolution of coronary artery spasm[12], the onset of the effect of nifedipine is slow. Verapamil is also effective for coronary artery spasm, because the drug easily causes bradycardia, and because it might be inappropriate to use in our case whose heart rate (HR) during coronary artery spasm was below 60 bpm. There has not been reported severe hypotension with incremental intravenous administration of low dose ISDN[3]. ISDN is also used during coronary arteriography for the resolution of coronary artery spasm exacting a general inhibition of smooth muscle contraction[4]. In this case, 7 min after the incremental intravenous administration of ISDN (total dose 3 mg) coronary artery spasm was resolved, and the ST-segment elevation was normalized without hemodynamic deterioration. We conclude that incremental intravenous ISDN administration was effective for the resolution of coronary artery spasm in this case.

The sudden ST segment elevation observed in this patient was considered to be caused by coronary artery spasm by the following reasons. A diagnosis of intraoperative coronary artery spasm is made only on the basis of transient or repetitive ST-segment elevation 2 mm or greater on ECG without an increase in heart rate or systolic blood pressure[8]. In this case, the ECG showed transient 7 mm ST-segment elevation without an antecedent rise in systolic blood pressure.

Past history of ischemic heart disease, the administration of sympathetic nerve stimulants, unstable anesthetic management and surgical stress have been reported to be triggering factors or causes of intraoperative coronary spasm[5–7]. There is no definite evidence whether anesthetic state or anesthetics induced coronary spasm. He had not only coronary risk factors of hypertension and smoking, but also 25% stenoses in segments 1 and 3 on coronary arteriogram. Furthermore, prior to admission he often had anginal attacks early in the morning. These findings are sufficient to diagnose vasospastic angina, and it was assumed that the patient already had spastic coronary arteries on arrival at the operating room before induction of anesthesia. We should have instituted continuous intravenous administration of ISDN or diltiazem before induction of anesthesia owing to the past history of

vasospastic angina. We used thiopental and diazepam as induction agents of general anesthesia, vecuronium as a muscle relaxant and topical intratracheal lidocaine. We have never recognized any of them as a potential agent for inducing coronary artery spasm in the literature. But immediately after intubation, rate pressure product increased to 12650, and the ECG showed ischemic-type ST segment depression. It is uncertain whether the transient ischemic episode was one of the triggering factors for the coronary artery spasm in this case, but we think anesthesia should have been induced with narcotics to prevent the hemodynamic deterioration.

In summary, we report a case showing an episode of myocardial ischemia caused by coronary artery spasm during general anesthesia. Incremental intravenous administration of ISDN attenuated the coronary artery spasm. We conclude that intravenous ISDN administration is effective for the resolution of coronary artery spasm during general anesthesia for non-cardiac surgery.

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