Hyperventilation Induced Coronary Artery Spasm during Anesthesia for Neurosurgery

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For patients with intracranial pathology, induced hyperventilation is recommended to reduce intracranial pressure during anesthesia[1,2]. But hyperventilation may cause angina and coronary artery spasm in some patients with or even without the presence of coronary artery disease[3–8]. We report a case in which coronary artery spasm, probably related with hyperventilation, occurred in a patient without previous history of coronary disease. This, to the best of our knowledge, has never been reported to occur in association with anesthesia for neurosurgery.

Report of a Case

A 59-year-old male patient (body weight 60 kg, height 165 cm) was scheduled for craniotomy for metastatic brain tumor originated from the lung under general anesthesia. Past medical history revealed no clinical episode except for the metastatic brain tumor. He was slightly drowsy due to intracranial hypertension. Physical findings and laboratory examinations disclosed no abnormalities. He had been medicated with decadron 6 mg·day^{-1} for reducing intracranial hypertension.

On the day of operation, the patient was given 10 mg diazepam orally, 90 min prior to induction of anesthesia. General anesthesia was induced with 300 mg of thiamylal sodium and 8 mg of pancuronium bromide. Manual moderate hyperventilation with 33% oxygen, 66% nitrous oxide and 2–3% enflurane was maintained by a mask for 5 min, and continued for further 1 min with 100% oxygen and 2% enflurane before tracheal intubation. Prior to tracheal intubation, 3 ml of 4% lidocaine was sprayed to tracheal mucosa. Tracheal intubation was uneventful with a slight change in arterial pressure (128/66 to 134/70 mmHg) and heart rate (72 to 76 bpm). The patient was ventilated with 33% oxygen, 66% nitrous oxide and 1–2% enflurane using an anesthesia ventilator (AIKA, Tokyo, Japan) with a tidal volume of 500 ml and respiratory rate of 15 bpm with stable arterial pressure and heart rate. Capnography (Normocap, Datex, Finland) indicated end-tidal carbon dioxide tension as 25 mmHg. Five minutes after tracheal intubation and mechanical ventilation, just before positioning of the patient abruptly electrocardiogram (ECG) displayed ventricular fibrillation, and mean arterial pressure fell to 50 mmHg. Cardiopulmonary resuscitation was immediately begun with chest compression, ventilation with 100% oxygen and intravenous administration of 60 mg of lidocaine. A minute later ECG returned to normal ventricular contraction with occa-

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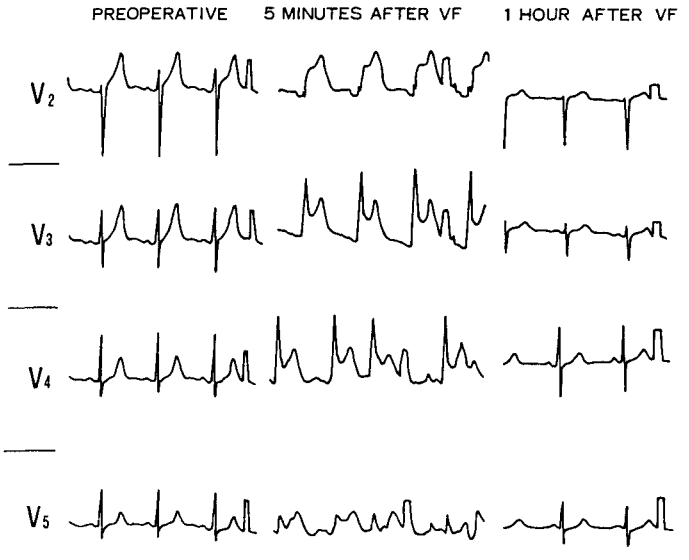

Fig. 1. Three phases of electrocardiogram indicate rapid changes in ST segment elevation.

sional premature ventricular contraction and 2 min after returned to the sinus rhythm. ECG monitor showed a remarkable elevation of ST segment on the modified II lead. The twelve leads ECG findings at that time were marked ST elevations in II, III, AVF and V3–V6 and the tentative diagnosis was anterior-inferior ischemia according to the cardiologist. Two $\mu g \cdot kg^{-1} \cdot min^{-1}$ of nitroglycerin and 1 $mg \cdot kg^{-1} \cdot min^{-1}$ of lidocaine were infused to control arrhythmia. ST segment elevation was returned to the baseline 1 hour later (fig.1).

Arterial blood gas and plasma electrolyte measurements at the time of ventricular fibrillation showed pH 7.56, Pa_{O_2} 350 mmHg, Pa_{CO_2} 28 mmHg, BE 1.2 $mEq \cdot l^{-1}$, Na 138 $mEq \cdot l^{-1}$, K 2.8 $mEq \cdot l^{-1}$ Ca 0.98 $mEq \cdot l^{-1}$. This hyperventilation was corrected by decreasing the respiratory rate to 10 bpm.

The surgery was canceled. The patient was transferred to the intensive care unit and his ECG was within a normal limit. Results of serum enzyme studies (SGOT, LDH, CPK) were normal, indicating absence of myocardial infarction. The follow up coronary angiogram showed a slight narrowing of the left anterior descending artery.

Discussion

Variant angina pectoris, which was first described as a clinical entity by Prinzmetal et al.[9], is diagnosed only electrocardiographically by the presence of a ST-segment elevation in an area supplied by major coronary artery. There may not be an associated history of chest pain. The probable cause of the syndrome is coronary artery spasm which may be exaggerated by hyperventilation[3–8], calcium injection[10] and alpha-adrenergic reaction[11]. Different from usual angina pectoris, Prinzmetal's variant angina occurs at rest or during the light phase of sleep with specific transient ST elevation[9]. Although several cases have been reported on the coronary spasm during anesthesia, we don't know a case of such an occurrence in patients during neurosurgery[13–15].

During anesthesia for neurosurgery, it is a common practice to reduce intracranial pressure by hyperventilation[1,2,10]. Recommended Pa_{CO_2} level by passive hyperventilation is between 25 to 30 mmHg[10].

Hyperventilation has been used during angiography to provoke coronary spasm[6]. Time sequence of ST elevation in the present case was similar to that reported in the previous study[6]. However the value of Pa_{CO_2} during coronary spasm in the present patient was lower than that of previously reported cases.

Exercise-induced myocardial ischemia in patients with fixed coronary stenosis results from increased myocardial oxygen demand secondary to a rise in blood pressure, heart rate and/or contractility. Oxygen delivery through the stenotic vessel fails to meet this augmented demands, and myocardial tissue ischemia results. In contrast, coronary vasospasm partially or totally reduces blood flow and oxygen delivery to the the affected myocardium[6]. Although an increase in heart rate and/or blood pressure precedes and causes effort-induced myocardial ischemia, no such hemodynamic changes indicate spasm-induced ischemia. ST segment changes and symptoms of acute left ventricular failure occur simultaneously and develop rapidly. Systemic arterial pressure and cardiac output fall, and left ventricular enddiastolic pressure increase[13–15].

The exact etiology of coronary spasm is still unclear, but Maseri and Chiercha[16] suggested that it may present local hypersensitivity to a normal physiological stimulus at certain times. This case had clinically insignificant mild coronary artery stenosis which might have produced vasoconstrictor activity[17]. Coronary spasm is due to strong contraction of coronary vascular smooth muscle cells triggered by an increase of intracellular ions; hyperventilation plus TRIS-buffer infusion are reported to induce coronary spasm by decreasing hydrogen ions which antagonize the action of calcium ions[3,10].

Administration of nitroglycerin promptly relieves the acute attack of coronary spasm, and calcium antagonists such as diltiazem, nifedipine and verapamil, which block the entry of calcium ions into coronary vascular smooth muscle cells and dilate large coronary arteries, prevent the occurrence of coronary spasm[6].

There is no definite evidence whether anesthetic state or anesthetics augment or relieve hyperventilation induced coronary spasm. The action of nitrous oxide on heart and peripheral circulation have been extensively investigated, and are controversial. Wilkowski et al.[18] indicated that sixty percent nitrous oxide produces constriction of epicardial coronary artery. Although normal coronary artery possesses considerable reserve capacity, and the constriction would have little impact on blood flow to the heart, there is no information as to the effects on coronary artery with slight stenosis.

In this case, we used thiamylal as an induction agent of general anesthesia, enflurane as a main inhalation anesthetics, pancuronium bromide 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.

The cardiac risk associated with noncardiac surgery is a clinically silent or apparently stable coronary artery disease that is unmasked by the stress of anesthesia and surgery. Since it is too hazardous and expensive to perform coronary angiography on all preoperative patients, in general, when the patient has very low cardiac risk, it might be permitted not to do any further invasive or non-invasive tests[19] before anesthesia and surgery.

Although the hemodynamics were recovered rapidly because cardiopulmonary resuscitation was started immediately after circulatory depression caused by ventricular fibrillation, yet recovery of ST elevation required about one hour in consequence of treatment including the reduction of minute ventilation to be normocarbia and nitroglycerin administration.

In summary, we suggest that even moderate hyperventilation may cause coronary spasm in patients without previous episode of coronary artery disease.

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