Case Report

Coronary Artery Spasm during Epidural Plus General Anesthesia

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Abstract: We present a case of coronary artery spasm during epidural plus general anesthesia. A 71-year-old man was scheduled for pancreatoduodenectomy. A continuous thoracic epidural block was performed at the Th 10–11 interspace and general anesthesia was maintained with enflurane and nitrous oxide in oxygen. Thirty minutes after the start of the operation, hypotension occurred suddenly and the ECG showed elevation of the ST segment, widening of the QRS complex and occasional ventricular premature beats. These changes resolved abruptly without treatment. The same episodes occurred both 1 hour and 30 min before the end of the operation, and were considered to be caused by coronary artery spasm. We used a continuous nitroglycerin infusion to prevent the coronary vasospasm. No further episodes of the coronary vasospasm occurred. It is uncertain that the coronary spasms in this patient were due to the hyperventilation or epidural anesthesia.

Key words: Anesthesia, Coronary spasm, Nitroglycerin

INTRODUCTION

During the operative period, myocardial ischemia is often caused by an increase in myocardial oxygen consumption in patients with coronary artery disease. In contrast to effort-induced ischemia, myocardial ischemia can also result from coronary artery spasm. The systemic hemodynamic pattern associated with coronary artery spasm differs from that associated with effort-induced ischemia. Coronary vasospasm occurs suddenly without a provocative increase in blood pressure or heart rate and may frequently provoke life-threatening dysrhythmias.

Kano et al.2) reviewed the 14 cases of coronary artery spasm under epidural or spinal anesthesia reported in the Japanese literature. However, a patient in whom coronary spasm occurred more than twice has seldom been reported. We present such a case of coronary artery spasm during epidural plus general anesthesia.

REPORT OF A CASE

A 71-year-old man was scheduled for pancreatoduodenectomy. He had experienced the first attack of angina pectoris 5 years ago, but had not experienced the pain for the past 1 year. He had not received medical treatment for this symptom. The chest X-ray and the electrocardiogram (ECG) at rest were normal. An exercise tolerance test revealed the depression of the ST segment in leads II, III and aVf in ECG. Results of enzyme studies were normal except for SGOT, SGPT, CPK and To-Bil (90 mIU, 106 mIU, 607 mIU, 4.0 mg/dl, respectively).
The patient was premedicated with hydroxyzine 25 mg and atropine 0.5 mg intramuscularly 30 min before arriving in the operating room. Intravenous and intra-arterial catheters were inserted and ECG lead V5 was monitored. A continuous thoracic epidural block was performed at the Th 10–11 interspace. Anesthesia was induced with intravenous thiamylal 250 mg while the patient breathed 100 per cent oxygen. Following loss of consciousness, succinylcholine 60 mg was administered, and the trachea was intubated. Anesthesia was maintained with enflurane and nitrous oxide in oxygen. Five millilitre of 0.25% bupivacaine was injected to produce an epidural block. Arterial pressure decreased from 130/80 to 102/66 mmHg over a 10-min period.

Thirty minutes after the start of operation, the ECG suddenly showed elevation of the ST segment, widening of the QRS complex and occasional ventricular premature contractions. The arterial pressure decreased to 65/39 mmHg (Fig. 1). These changes resolved abruptly without treatment 3 minutes later. As the blood gas analysis showed respiratory alkalosis (pH

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Fig. 1. ECG and blood pressure changes during coronary artery spasm. ECG showed elevation of the ST segment and widening of the QRS complex. The arterial pressure decreased to 65/39 mmHg.
Coronary spasm during anesthesia

7.53, PaCO₂ 24.6, PaO₂ 186). Ventilation was controlled to maintain PaCO₂ between 35 and 40 torr. However, the same episode occurred 1 hour before the end of operation. This episode also was not preceded by any rise in heart rate or arterial blood pressure. When the third similar event occurred, a continuous nitroglycerin infusion was administered at a rate of 1.0 μg/kg/min through the line for nitroglycerin. The arterial pressure returned to normal (BP 120/80 mmHg, HR 76 beats/min). After the operation, results of enzyme studies (SGOT, LDH, CPK) were almost identical to those before the operation. As for LDH isoenzymes, LDH₁/LDH₂ ratio was less than 1, and there was no difference between the ECG before and after the operation. These findings did not indicate myocardial infarction. No further episodes of ST segment elevation or dysrhythmias occurred, and the patient was discharged from the hospital.

**Discussion**

Three paroxysmal episodes of myocardial ischemia observed during epidural plus general anesthesia in this patient were probably caused by coronary artery spasm, as the ST segment elevation was not preceded by an increase in either heart rate or arterial pressure. Effort-induced myocardial ischemia in patients with fixed coronary stenoses occurs when myocardial oxygen demand is increased by an increase in heart rate, blood pressure, or contractility. In contrast, coronary vasospasm decreases blood flow and oxygen delivery to the myocardium, and the onset of spasm-induced ischemia is sudden and without warning⁹. Systemic arterial pressure and cardiac output fall, and left ventricular end-diastolic pressure increases. However, most episodes resolve within 2–5 min⁹. If vasospasm is marked and prolonged it can lead to an acute myocardial infarction⁹.

We can only speculate as to the origin of the vasospasm during anesthesia. First, hyperventilation can be the cause. The vasoconstriction occurs if the hydrogen ion concentration decreases by hyperventilation¹². There are reports that hyperventilation causes coronary vasoconstriction in both normal subjects¹¹ and patients with stable exertional angina pectoris⁶. The first attack in this patient may have been due to the hyperventilation. Second, epidural anesthesia may be the cause⁴. Above the level of sympathetic blockade, compensatory vasoconstriction may occur, presumably in an effort to offset the reduction in peripheral resistance caused by the block. This reflex sympathetic activity may involve the cardiac sympathetic nerves causing coronary vasoconstriction. In contrast to this view, thoracic epidural block produces coronary vasodilation and decreased myocardial oxygen consumption¹⁰. Sympathectomy by thoracic epidural anesthesia redistributes coronary blood flow to the endocardium with an improvement in the endocardial/epicardial ratio, both infarcted and uninfarcted hearts³. Kano *et al.*² reviewed the cases of coronary artery spasm under epidural or spinal anesthesia reported in the Japanese literature, classified them into two groups according to the existence of denervation of the cardiac sympathetic nerves, and discussed the possible factors of triggering coronary artery spasm in each group. Third, vagal reflex due to surgical stimulation may be the cause. However, bradycardia and hypotension were not seen before the attack in this patient.

We used a continuous nitroglycerin infusion to prevent the coronary vasospasm. The efficacy of nitrates in preventing attacks of variant angina has been recognized
since Prinzmetal’s first report9). Hypotension accompanying myocardial ischemia suggests cautious administration of a vasodilator drug such as nitroglycerin, however, aggressive treatment is required in cases of ischemia caused by coronary spasm. Doses of nitroglycerin should be adjusted to avoid tachycardia or hypotension.

Although we did not administer calcium channel blocking drugs, they are also coronary vasodilators that decrease the frequency of variant angina attacks in clinical situations1,8).

In summary, we encountered a patient undergoing surgery and epidural plus general anesthesia who had three times episodes of myocardial ischemia probably caused by coronary artery spasm. The intravenous nitroglycerin was useful in preventing coronary spasm during anesthesia.

References


硬膜外と全身麻酔の併用下に起きた冠動脈スパスムの一例

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症例は71歳の男性で脾頭十二指腸切除術が施行された。麻酔は硬膜外カテーテルを陰部の 10-11に挿入し、エンフルエンの全身麻酔とした。手術開始30分後に、突然血圧が低下し、心電図は ST の上昇、幅の広い QRS、心室性期外収縮を示した。これらは約1分後に自然に軽快した。同じような発作が手術終了30分と1時間前に起こった。冠動脈スパスムが原因と考えられたので、3度目の発作の時にニトログリセリンの静注を行った。以後発作は退院時までおこらなかった。この症例の冠動脈スパスムの原因として、術中の過換気や硬膜外麻酔が考えられるが、確定はできない。

キーワード 冠動脈スパスム、麻酔、ニトログリセリン