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Intraoperative coronary artery spasm with hemorrhagic shock state

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Introduction

The balance between the myocardial oxygen supply and demand is the single most important factor in the system controlling coronary circulation. Since oxygen demand by the heart determines coronary blood flow, a perioperative increase in myocardial oxygen demand often induces myocardial ischemia in patients with fixed atheromatous obstruction of the coronary arteries [1]. In contrast, coronary artery spasm, which is not preceded by significant changes in the factors influencing myocardial oxygen demand, is recognized as an important cause of decreased myocardial blood flow [2].

Myocardial oxygen supply is also decreased in severe anemia with hypovolemia, such as during a state of hemorrhagic shock [3]. Accordingly, coronary artery spasm combined with hemorrhagic shock induces severe myocardial ischemia, which may be life-threatening even in patients without ischemic heart disease.

We present a patient in whom coronary artery spasm during the state of hemorrhagic shock resulted in ventricular tachycardia during surgery. The patient was successfully treated without myocardial infarction.

Case report

A 61-year-old man, 59 kg, 164 cm, with primary hepatic carcinoma, was scheduled for right lobectomy of the liver. The patient had no history of hypertension or angina pectoris. Preoperative electrocardiogram (ECG) was normal, and a chest X-ray showed no abnormality. Laboratory data revealed the following: WBC, 6300/mm^3; hematocrit, 36.2%; platelets, 122 000/mm^3; prothrombin time, 13.2 s (control below 13.0 s); partial thromboplastin time, 31 s (control 32 s); bleeding time, 120 s (control below 300 s); serum sodium level, 138 mEq/L; serum potassium level, 3.8 mEq/L; serum aspartate aminotransferase (AST), 68 U; serum alanine aminotransferase (ALS), 88 U; serum lactate dehydrogenase (LDH), 382 U. (control up to 500 U.); serum cholinesterase, 0.88 ΔpH (normal range of 0.6–1.2 ΔpH).

The patient was premedicated with atropine 0.5 mg and midazolam 3.0 mg i.m. 30 min before the patient was brought into the operating room (OR). In the OR, central venous and intraarterial catheters were inserted and ECG leads were attached. Central venous and arterial pressures, ECG, percutaneous oxygen saturation, and rectal temperature were simultaneously monitored. The initial blood pressure (BP) and heart rate (HR) were 120/82 mmHg and 80 bpm with normal sinus rhythms, respectively, and rectal temperature was 36.2°C.

Anesthesia was induced by intravenous administration of fentanyl 500μg and midazolam 5 mg with 2.0% isoflurane in nitrous oxide (50%) and oxygen (50%) and the trachea was intubated by pancuronium (8 mg i.v.). Anesthesia was maintained with 1.0%–2.5% isoflurane in nitrous oxide (50%) and oxygen (50%). Ventilation was controlled to maintain $Paco_2$ between 33 and 38 mmHg. Before the surgery, BP and HR stabilized at 110/60 mmHg and 70 bpm with normal sinus rhythms (Fig. 1a).

One hour after the start of surgery, the right hepatic vein was accidentally ruptured. Approximately 3000 ml of blood was lost in 10 min, and the bleeding could not be controlled. Systolic BP dropped to 30 mmHg, and the HR was 130 bpm; however, there was no arrhythmia

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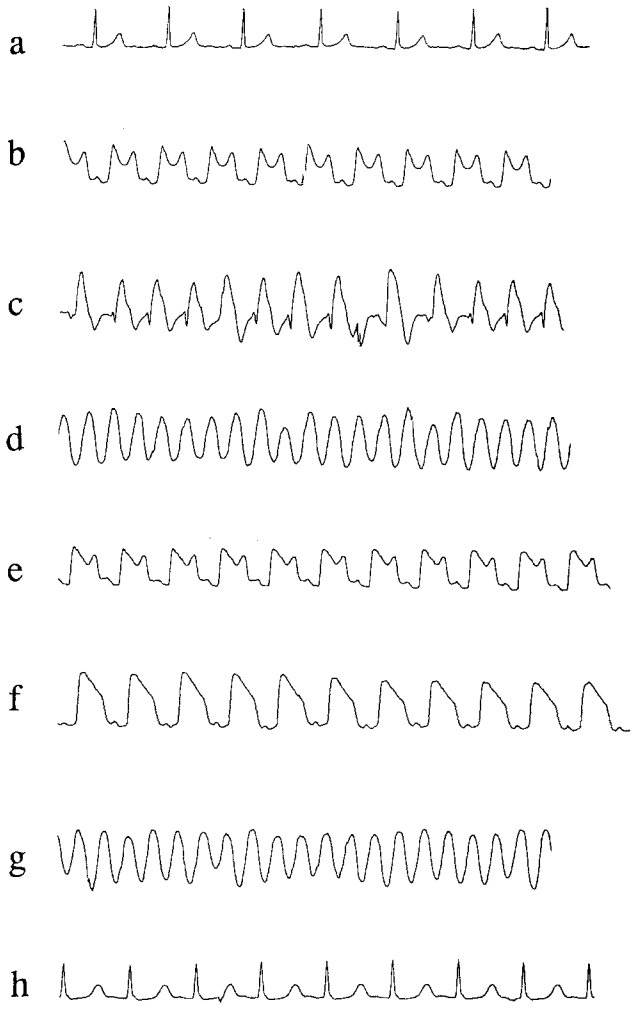

Fig. 1a–h. Electrocardiograms before and during surgery. **a** Before surgery. **b–d** ST segment elevation and ventricular tachycardia. **e** After administration of lidocaine 100 mg and nitroglycerine 0.3 $\mu g \cdot kg \cdot min^{-1}$. **f–g** ST segment elevation and recurrent ventricular tachycardia. **h** After administrations of nicardipine 2 mg and nitroglycerine 0.8 $\mu g \cdot kg \cdot min^{-1}$

or change in the ST segment. Administration of isoflurane and nitrous oxide were immediately discontinued, and the patient was given 100% oxygen. After a bolus injection of adrenaline 0.05 mg i.v. continuous administration of dopamine 10 μg/kg/min i.v. was started. One thousand milliliters of 5% albumin solution was rapidly administered, and was then followed by the administration of 10 units of packed red cells and 3 units of fresh frozen plasma which were derived from 400 ml of blood. After these administrations, hematocrit was 20% and central venous pressure (CVP) was 2 cmH_2O.

At this time, the arterial blood gases showed the following: pH, 7.34; $Paco_2$, 33 mmHg, Pao_2, 348 mmHg; base excess, −6.8 mEq/L. One hundred milliliters of 7% $NaHCO_3$ and additional transfusions of 8 units of red blood cells and 2 units of fresh frozen plasma were administered as well as 1000 ml of 5% albumin containing solution. BP increased to 50/20 mmHg, and HR decreased to 130 bpm, with 3 cmH_2O of CVP; however, the bleeding was not yet under control and blood transfusion was continued. Thirty minutes later, BP and HR were 70/30 mmHg and 110 bpm, and the bleeding was gradually reduced. Until this time, approximately 20 units of packed red blood cells, 10 units of frozen fresh plasma and 2000 ml of 5% albumin containing solution were administered. Fentanyl 200 μg i.v. and midazolam 2.5 mg i.v. were administered, then anesthesia was maintained with isoflurane 0.6%–1.0% in nitrous oxide (50%) and oxygen (50%).

Ten minutes later, BP was 70/40 mmHg with 5 cmH_2O) of CVP; however, ST segment elevation was noted on the monitor lead (Fig. 1b,c). The packed red blood cells and 5% albumin solution were rapidly administered intravenously and nitroglycerine 0.3 μg/kg/min i.v. was continuously infused; however, 10 min later, the ST elevation was followed by recurrent ventricular tachycardia (Fig. 1d). Anesthesia was discontinued and lidocaine 100 mg i.v. given. At this time, rectal temperature was 35.1°C. The Pao_2 was 148 mmHg, the $Paco_2$ was 35 mmHg and the blood pH was 7.42; hematocrit was 23%, plasma sodium was 136 mEq/L, plasma potassium was 3.2 mEq/L, and plasma calcium was 3.3 mg/dl. The ventricular tachycardia was successfully treated within 3 min (Fig. 1e,f). However, marked ST elevation persisted, and was again followed by ventricular tachycardia (Fig. 1g).

We diagnosed this as myocardial ischemia caused by coronary artery spasm. Intravenous injection of nicardipine 2 mg was followed by administration of nitroglycerin 0.8 μg/kg/min i.v. Soon after the administrations, ECG showed normal sinus rhythms (Fig. 1h), and BP was 80/40 mmHg and HR was 92 bpm. Fentanyl 500 μg and midazolam 2.5 mg were additionally given and anesthesia was maintained with isoflurane (1.2%–2.0%) in nitrous oxide (50%) and oxygen (50%). BP and HR were maintained at 100/60 mmHg and 80 bpm. No further episodes occurred and surgery was completed without further complications.

After surgery, the patient was transferred to the intensive care unit with the endotracheal tube in place and was mechanically ventilated. No abnormal findings were detected on a 12-lead ECG recorded immediately after the operation. Six hours after surgery, the patient was awake to obey verbal commands; 16 h later, he was able to be extubated. Postoperative laboratory tests did not reveal any abnormalities in enzymes, such as LDH-1 and CPK-MB. There were no abnormal neurological findings in the patient and the electroencephalogram also showed no abnormality. No similar episodes in the ECG were observed throughout the postoperative period and the patient completely recovered. One month

after the surgery, Holter monitoring and exercise ECGs showed no arrhythmias or ST segment changes.

Discussion

Perioperative coronary artery spasm provokes life-threatening dysrhythmias and marked changes in left ventricular mechanical performance [4]. Many factors are involved in the occurrence of perioperative coronary artery spasm [5–8]. However, to our knowledge, there have been no reports describing the perioperative occurrence of coronary artery spasm combined with the state of hemorrhagic shock, probably because of its high mortality; of course, under the circumstances, it may be difficult to trace myocardial ischemia to coronary artery spasm.

We first interpreted this patient's ST elevation as the result of myocardial ischemia due to severe hypovolemic shock from the additional large loss of blood; however, the hemorrhage had reduced, and the patient had shown signs of recovering from the state of hemorrhagic shock when the changes abruptly occurred in the ECG. Thus, we re-interpreted it as coronary artery spasm combined with hypovolemic shock. Although it is known that systemic hypotension induces intraoperative coronary spasm, it remains unclear that hypotension and/or hypovolemia due to the hemorrhagic shock state per se induced coronary spasm in the present patient, because it occurred in the recovering period from the shock state. The contribution of sympathetic nerve stimulation secondary to the shock state and cold from the rapid blood transfusion might have been involved in the coronary artery spasm in the present patient; however, it may be necessary to take other sources into consideration.

Thrombin, which is readily activated by tissue damage and bleeding, has been shown to be a potent vasoconstrictor of coronary arteries [9] and may induce coronary spasm; and chemical mediators, including histamine, serotonin and thromboxane A2, are known to be involved in the pathogenesis of coronary spasm associated with allergic reaction [10,11]. Allergic reactions also occur in 3% of all transfusions, including anaphylaxis, though this is rather rare [12]. In the present patient, about 20 units of red blood cells and 8 units of fresh frozen plasma were administered for the bleeding until the ST segment elevated. Thus, bleeding and large blood transfusion might have caused the patient's coronary arterial spasm.

Since many factors can be involved in the pathogenesis of coronary arterial spasm in the clinical course and the treatment of hemorrhagic shock state, it is difficult to determine precisely the cause of the spasm. Apart from the pathogenesis, as coronary arterial spasm further aggravates myocardial ischemia in the hemorrhagic shock state, rapid treatment is required. Several drugs that have had extensive trial outside Japan as coronary arterial vasodilators and antiarrhythmatic agents may prove to be of great importance in treating coronary artery spasm [2]. The calcium antagonists verapamil, diltiazem, nifedipine, and perhexilline have all been reported to be effective in treating coronary arterial spasm [2,13–15]. We chose nicardipine, also a calcium antagonist, because it is a more potent dilator of coronary arteries than these calcium antagonists [16]. The effect of nicardipine was dramatic in the present patient. Although nitroglycerin is also known to be effective in reversing coronary artery spasm [2], the infusion at a rate of $0.3\ \mu g \cdot kg \cdot min^{-1}$ did not show any effect. Blood levels may not have been high enough to prevent coronary artery spasm in the present patient. The infusion of nitroglycerin at $0.8\ \mu g \cdot kg \cdot min^{-1}$ seems to have been effective in the present patient. We were afraid of a decrease in BP after the administration of nicardipine and nitroglycerin; however, BP was maintained, probably as a result of a strengthened left ventricular contraction after the coronary artery spasm had been reversed.

In summary, we have described a patient undergoing surgery in whom coronary arterial spasm occurred in the state of hemorrhagic shock. The patient had no history of angina pectoris or hypertension. The ST segment abruptly rose during the state of hemorrhagic shock. A rapid and effective response to intravenous nicardipine 2 mg and nitroglycerin $0.8\ \mu g \cdot kg \cdot min^{-1}$ was observed, and the patient was successfully treated without myocardial infarction. In this patient, coronary arterial spasm might have occurred following the state of shock per se or secondary to this condition.

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