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Electrocardiographic ST segment elevation during dopamine infusion for massive bleeding

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Introduction

Dopamine produces coronary artery vasodilation in proportion to the increased myocardial oxygen demand [1]. However, tachycardia may occur with dopamine, especially at higher doses, producing increased oxygen demand. Coronary heart disease produces symptoms when myocardial oxygen demand exceeds supply, and local or systemic regulatory mechanisms fail to correct perfusion imbalance. In addition, dopamine has been shown to provoke coronary spasm [2].

All ST segment elevations greater than 1.0 mm are considered significant [3]. This is a manifestation of more severe myocardial ischemia reflecting transmural, rather than subendocardial, ischemia. In patients with Prinzmetal angina (coronary spasm), the ST segments are also elevated with pain [3].

This report describes a case in which marked ST elevation was seen after large doses of dopamine infusion for severe hypotension due to massive bleeding.

Case report

A 50-year-old man suffering from hepatocellular carcinoma was scheduled for right lobectomy of the liver. At the time of surgery, his physical examination and preoperative laboratory tests were normal. His hematocrit was 44%, and his platelet count was $220 \times 10^9 l^{-1}$.

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An epidural catheter was placed via Th8–Th9 before induction of anesthesia for postoperative pain relief.

He received, as routine premedication, 0.5 mg atropine sulfate and 2 mg midazolam intramuscularly. After appropriate monitors had been placed, including a cannula in his left radial artery for continuous blood pressure monitoring, general anesthesia was induced with intravenous 200 mg thiopental, 100 μg fentanyl, and 7 mg vecuronium bromide. After tracheal intubation, anesthesia was maintained with isoflurane, nitrous oxide, fentanyl, and vecuronium bromide. A central venous catheter was placed via the right subclavian vein.

About 3 h after the beginning of surgery, the patient suffered unexpected massive bleeding from the inferior vena cava. Before the massive bleeding, arterial blood pressure had ranged from 82/52 to 145/85 mmHg and pulse rates from 58 to 90 beats·min^{-1}. His blood pressure fell from 112/61 to 42/25 mmHg and central venous pressure fell from 5 to 0 mmHg (Fig. 1). The ECG showed a depressed ST segment (Fig. 2), indicating myocardial ischemia. With rapid transfusion of concentrated red blood cells and plasma constituents, and continuous dopamine infusion of 20 μg·kg^{-1}·min^{-1}, the blood pressure was restored to 82/40 mmHg and the heart rate was 110 beats·min^{-1}. However, the ECG showed ST elevation in leads I, II, III, aVF, aVL, and V, and soon after complete AV block ensued (Fig. 2). Bolus and continuous infusion of isosorbide dinitrate, reduction the speed of infusion of dopamine from 20 to 5 μg·kg^{-1}·min^{-1}, and additional blood transfusion alleviated these changes after 20 min (Fig. 2). During the surgery, lactated Ringer's solution, 2100 ml, and 5% albumin, 5000 ml, were infused intravenonoly, and 20 units of fresh frozen plasma and 18 irradiated units of concentrated red blood cells were transfused. Blood loss was 4800 ml. The hematocrit was 25% and the platelet count was $67 \times 10^9 l^{-1}$ at the end of surgery.

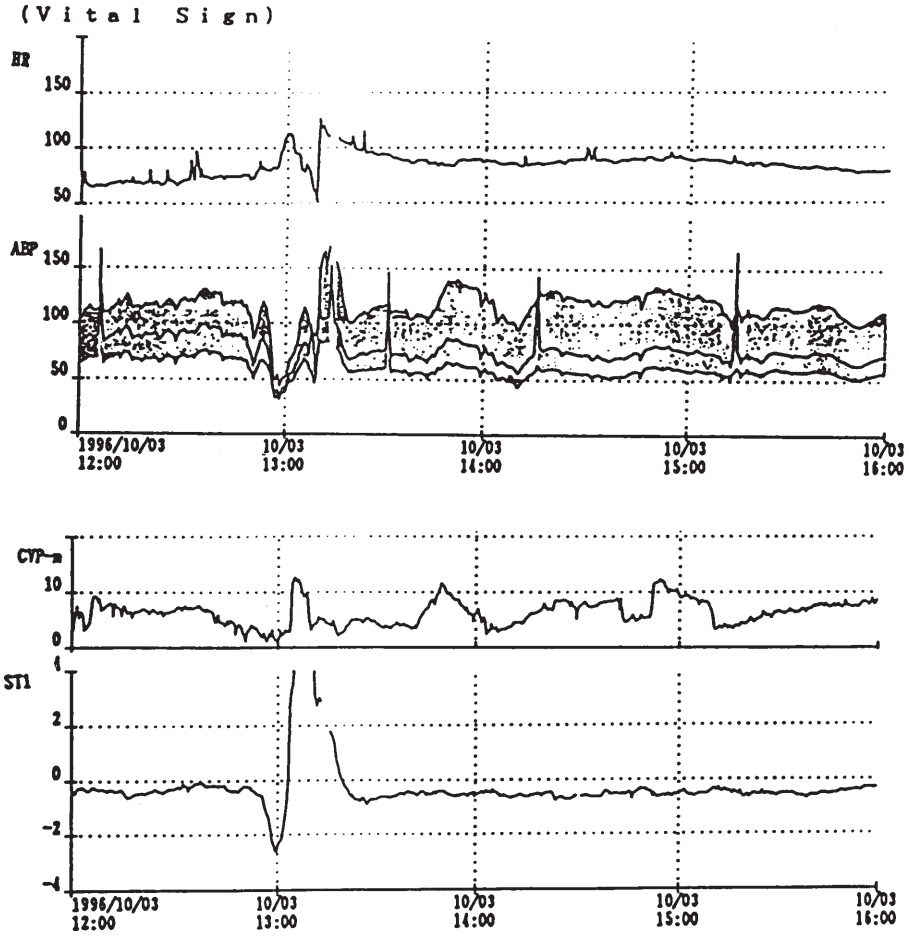

Fig. 1. Vital signs of patient. *HR*, Heart rate (beats·min^{-1}); *ABP*, arterial blood pressure in radial artery (mmHg); *CVP-m*, mean central venous pressure (mmHg); *ST1*, ST segment in ECG lead I (mm)

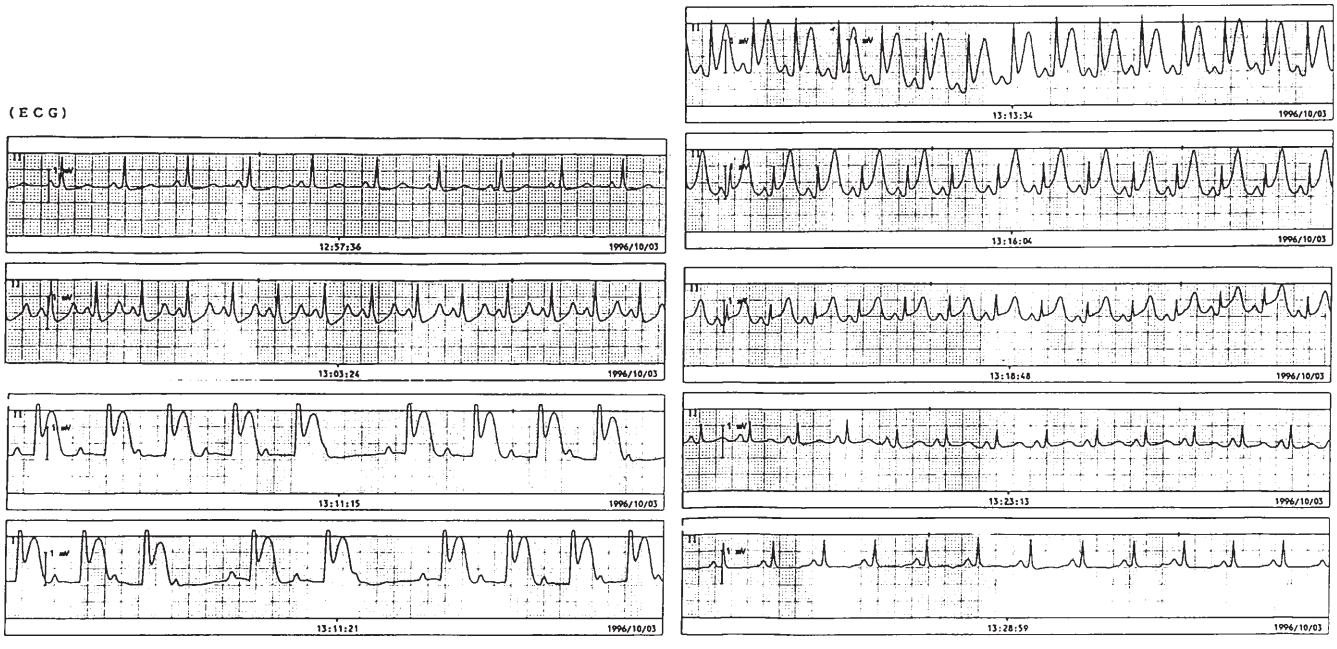

Fig. 2. Appearance and disappearance of ST elevation and complete AV block in ECG leads II. Paper speed, 25 mm·s^{-1}; 1 mV = 1 cm

The patient was subsequently transported to the intensive care unit and remained hemodynamically stable. There was no significant postoperative bleeding. Recovery proceeded uneventfully. The trachea was extubated the following morning. The ECG did not show development of Q waves or changes in QRS. The subsequent postoperative course was uneventful.

Discussion

Ruiz et al. [4] reported that in spite of its beneficial effects in shock, patients with marked hypoperfusion and increased lactate levels usually do not respond to therapy with dopamine. In such cases, cardiovascular reserve is totally exhausted and the heart appears unresponsive to any type of sympathomimetic support. However, in order to avoid hypoperfusion, sympathomimetic support in the early phase of massive bleeding seems effective.

In the present case, the sudden onset, prominent magnitude, and brief duration of the ST elevation may be related to a transient coronary vasoconstriction induced by the large doses of dopamine infusion. The effects of dopamine on the arteries are different, depending on the dose and receptor population. Its administration can cause vasodilation by stimulation of dopaminergic receptors [5] and vasoconstriction by stimulation of alpha-adrenergic and serotonergic receptors [6]. Crea et al. [2] assessed the ability of dopamine to provoke coronary spasm in 18 patients with active vasospastic angina in whom this amine was infused at rates of 5, 10, and 15 $\mu g \cdot kg^{-1} \cdot min^{-1}$ for periods of 5 min each. They reported that in nine patients, dopamine caused angina and ischemic electrocardiographic changes suggestive of coronary spasm: ST segment elevation in six patients and ST segment depression in the absence of important coronary stenoses in the remaining three.

In coronary spasm, nitrates abolish or prevent myocardial ischemia exclusively by exerting a direct vasodilating effect on the spastic coronary arteries [3]. Isosorbide dinitrate effectively ameliorated ST elevation in the present case.

Another possibility is severe transmural, rather than subendocardial, ischemia due to severe hypotensions. Myocardial oxygen demand exceeds supply, and systemic regulatory mechanisms fail to correct perfusion imbalance because of massive bleeding. Although dopamine may produce coronary artery vasodilation in proportion to the increased myocardial oxygen demand [1], tachycardia occurs with dopamine, especially at higher doses, producing increased oxygen demand. In addition, because of the leftward shift in the oxygen dissociation curve, tissue hypoxia may develop from infusion of stored blood. The oxygen affinity of hemoglobin may be changed during preservation, making it difficult for hemoglobin to release oxygen to the tissues immediately after transfusion [7]. In this case, dopamine-induced tachycardia may have worsened the energy balance under the hypoperfusional condition.

In conclusion, marked ST elevation was seen after large doses of dopamine infusion for severe hypotension due to massive bleeding. The cause of ST elevation may be coronary spasm or transmural ischemia due to perfusion imbalance. However, large doses of dopamine do not seem to be contraindicated in massive bleeding to buy time for transfusion.

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