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Coronary artery spasm induced by respiratory alkalosis

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Introduction

Induction of coronary artery spasm by alkalosis is well known [1,2]. However, except for open heart surgery, very few cases have been related to intraoperative respiratory alkalosis [3]. We report herein a case of coronary arterial spasm probably induced by a marked respiratory alkalosis due to a ventilator malfunction.

Case report

A 58-year-old man, 163 cm tall and weighing 65 kg, presented with a pseudofracture of the right forearm in 1988. Fixation with bone transplantation was planned. He had no past history of hypertension or angina pectoris. Physical examination and laboratory tests were unremarkable.

Course of anesthesia

The patient was premedicated with atropine 0.5 mg and hydroxyzine 50 mg 30 min prior to induction of anesthesia. At induction of anesthesia, no abnormalities were noted in heart rate, blood pressure, or ECG. Anesthesia was induced by intravenous administration of fentanyl 200 μg followed by thiamylal 250 mg. Endotracheal intubation was performed with pancuronium 6 mg IV. Anesthesia was maintained with 3 l·min^{-1} nitrous oxide, 3 l·min^{-1} oxygen, and 1.5% enflurane. The ventilator was set at a tidal volume of 550 ml and a rate of 11 breaths·min^{-1}. About 10 min later (10 min after intubation), the blood pressure fell from 139/80 to 99/55 mmHg. At the same time, a nodal rhythm appeared in the ECG (Fig. 1a.). Blood pressure gradually recovered to 110/60 mmHg in response to a fluid load. At about 20 min after induction of anesthesia, ST segment elevation was noted in the ECG (Fig. 1b.), followed 2 min later by another fall in blood pressure to 95/60 mmHg and a marked ST elevation (Fig. 1c.). Although blood pressure recovered a few minutes later, the ST elevation persisted (Fig. 1d.). Coronary artery spasm was strongly suspected and intravenous nitroglycerin (NTG) was administered at a rate of 1.0 μg·kg^{-1}·min^{-1}. Thereafter, the ST segment returned close to the baseline (Fig. 1e.), but the elevation did not completely normalize (Fig. 1f.). Respiratory alkalosis was confirmed by an arterial blood gas analysis, demonstrating a pH of 7.548 and $Paco_2$ of 22.9 mmHg. The ventilatory setting was changed to 500 ml, 10 breath·min^{-1}, but the respiratory alkalosis still persisted. Another blood gas analysis 30 min later showed a pH of 7.567 and $Paco_2$ of 21.7 mmHg. A ventilator malfunction was suspected and manual respiration was performed with resolution of the alkalosis to pH of 7.393 and $Paco_2$ of 38.3 mmHg, with disappearance of ECG abnormalities (Fig. 1g.). Surgery was started after NTG administration when the circulatory dynamics became more stable. Anesthesia was maintained with nitrous oxide-oxygen-enflurane (concentration of enflurane, 1.0%–2.0%) with intermittent administration of fentanyl (total doses 600 μg) and continuous administration of 0.5–1.0 μg·kg^{-1}·min^{-1} NTG. Nifedipine 10 mg was administered intranasally for hypertension at the end of surgery. No ECG abnormalities recurred and the patient left the operating room. No ECG abnormalities appeared in the subsequent course and the patient was discharged. Although coronary arteriography was recommended, it was not performed due to the patient's refusal.

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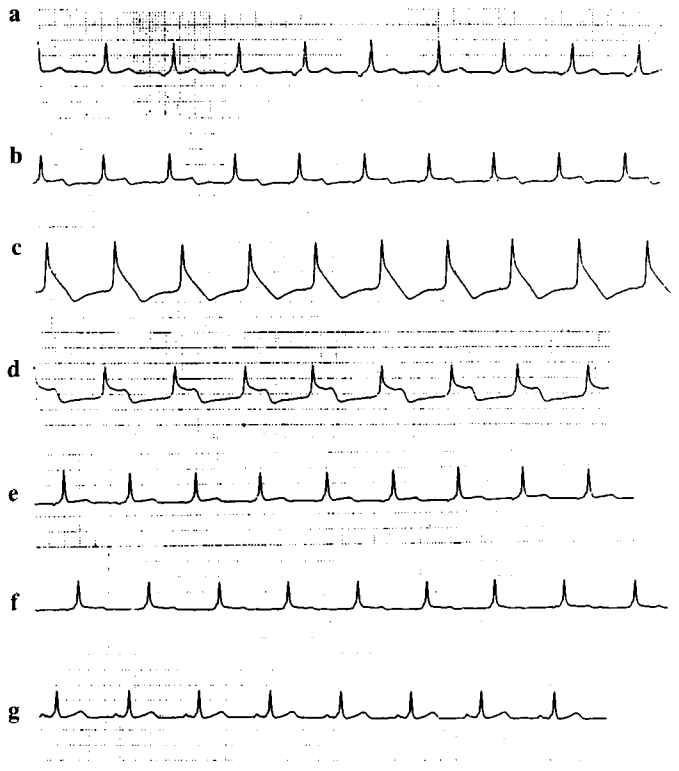

Fig. 1a–g. Changes of electrocardiogram during course of anesthesia

Discussion

In this patient, the ST segment elevation appeared after induction of anesthesia, without marked hemodynamic changes. NTG administration only partially corrected the ST elevation. After correction of respiratory alkalosis caused by hyperventilation, the ECG abnormalities did not recur. These findings strongly suggest that coronary artery spasm in this patient was induced by respiratory alkalosis.

Coronary artery spasm frequently occurs due to sympathetic nervous system stimulation and the resulting imbalance between the sympathetic and parasympathetic nervous systems [4–6]. In the present case, the ST elevation occurred 20 min after endotracheal intubation during nitrous oxide-oxygen-enflurane anesthesia but prior to the skin incision. Since reduction in blood pressure after induction of anesthesia was mild and returned to normal after a bolus of lactated Ringer's solution, it is unlikely that the increased sympathetic tone was responsible. Acetylcholine (Ach) dilates blood vessels in the presence of normal vascular endothelial cells, and contracts them when the endothelial cells are injured [7], and its injection into the coronary artery causes vasospasm [8]. Although secretion of Ach or increased parasympathetic tone could possibly induce coronary artery spasm, increased parasympathetic tone is very unlikely in the present patient.

Reports on the induction of coronary artery spasm by respiratory alkalosis during surgery are rare except during open heart surgery [3]. The incomplete recovery of the ST elevation by NTG administration and full recovery by correction of respiratory alkalosis strongly suggests that respiratory alkalosis was the main cause of the ST elevation in the case.

Respiratory alkalosis readily induces intracellular alkalosis because of CO_2 passing through the cell membrane. As the result, Ca^{++} inflow into the intracellular space is facilitated and the Ca^{++} sensitivity of the contractile protein is augmented [9–11], inducing an increase of coronary vascular resistance or coronary artery spasm [1,2,12–14]. Alkalosis, however, does not always induce coronary artery spasm, and requires some additional mechanism. In general, coronary artery spasm occurs in the presence of organic changes in the coronary artery [15]. Postoperative coronary arteriography in coronary artery spasm during anesthesia, however, sometimes fails to reveal organic changes. Recently, vascular endothelial cells have been reperted to be linked to the production of relaxing and contracting factors [7,16]. Even in the absence of organic changes in the artery, coronary artery spasms may occur when endothelial cells are injured or when endothelial function is augmented [16–19]. No past history of angina pectoris or other circulatory or metabolic disease was found in our patient. The presence or absence of organic changes of the coronary artery, and the reproducibility of the spasm should have been evaluated. However, it was not possible to perform coronary arteriography due to the patient's refusal.

In the present case, occurrence of marked respiratory alkalosis was unexpected, because the setting of tidal volume and respiratory frequency was proper. The result of blood gas analysis after ST elevation revealed marked hyperventilation. Despite subsequent change of the ventilating condition $Paco_2$ did not rise, and it was not until the beginning of manual artificial ventilation that alkalosis improved. Examination of the ventilator after anesthesia revealed leakage in the bellows, which probably caused hyperventilation. Our case suggests that monitoring of the volume of ventilation and end-tidal CO_2 concentration might have prevented the coronary artery spasm.

In summary, we reported an induction of coronary artery spasm induced by hyperventilation during anesthesia in a patient without any history of angina pectoris.

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