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Recurrent ST-segment elevation on ECG and ventricular tachycardia during neurosurgical anesthesia

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Abstract
This article reports an unusual case of repeated intraoperative myocardial ischemia and ventricular arrhythmia during neurosurgical anesthesia. The presentation was clinically diagnosed as coronary spasm after successful resuscitation. Intraoperative prostaglandin E_1 and β-adrenergic blockade, as well as vagal stimulation due to surgical manipulation, may have contributed to the episode.

Key words Coronary spasm · Anesthesia · Neurosurgical · Prostaglandin E_1 · Propranolol

Introduction

Coronary vasospasm may occur intraoperatively and cause serious ventricular arrhythmia and hemodynamic instability [1,2]. However, recurrent episodes of intraoperative coronary spasm in one patient during a single procedure is very rare. We present a case of multiple intraoperative episodes of ST change and ventricular tachycardia in a neurosurgical patient. These episodes were clinically diagnosed as coronary spasm, and the administration of a β-blocker and prostaglandin E_1 for deliberate hypotension may have been involved in the pathophysiology of the recurrent symptoms.

Case report

A 60-year-old, 54-kg, 157-cm-tall woman underwent neck clipping of an unruptured cerebral aneurysm. She had already undergone neck clipping of a ruptured cerebral aneurysm 10 years prior to this procedure, without any neurological sequelae. She had no history of coronary artery disease, and all the preoperative tests, including chest radiograph and ECG, revealed no abnormalities. Premedication consisted of oral famotidine and intramuscular meperidine, midazolam, and atropine sulfate. General anesthesia was induced with intravenous propofol and fentanyl, supplemented with vecuronium and maintained with continuous infusion of propofol and inhaled N_2O-O_2 (fractional inspired oxygen; $F_{I_{O_2}} = 0.33$). The maintenance dose of propofol was 4 $mg \cdot kg^{-1} \cdot h^{-1}$. The patient was mechanically ventilated to maintain Pa_{CO_2} at 30 to 35 mmHg. Before incision, administration of intravenous propranolol (0.6 mg) and continuous infusion of prostaglandin E_1, at a rate of 0.03 $\mu g \cdot kg^{-1} \cdot min^{-1}$, were started for deliberate hypotension. Twenty minutes after incision, a brief episode of bradycardia (heart rate [HR], 45 bpm), atrioventricular (AV) block, ST elevation, and T wave inversion with hypotension (arterial pressure, 74/48 mmHg) was noted (Fig. 1, trace 1A). Two minutes later, these changes disappeared, and only ST depression persisted (Fig. 1, trace 1B). After another minute, the ECG spontaneously returned to normal (Fig. 1, trace 1C). At this time, the HR was 74 bpm, and the blood pressure was 94/56 mmHg. The attending anesthesiologist diagnosed this episode as coronary vasospasm, and a transdermal isosorbide dinitrate patch was applied as a prophylactic measure. Prostaglandin E_1 administration was temporarily stopped and then restarted at the same dose 20 min later when microscopic manipulation was started. Eighty minutes after the first episode and during the microscopic manipulation of cerebral aneurysm, significant ST elevation and premature ventricular contractions were noted (Fig. 1, trace 2). At this time, the HR was 84 bpm, and the blood pressure was 89/46 mmHg. One minute later, ventricular tachycardia was noted on the ECG and was successfully treated with 60 mg of intravenous lidocaine. Two minutes after this event, the ECG returned to normal following transient ECG evidence of ST depression and T wave inversion. After this

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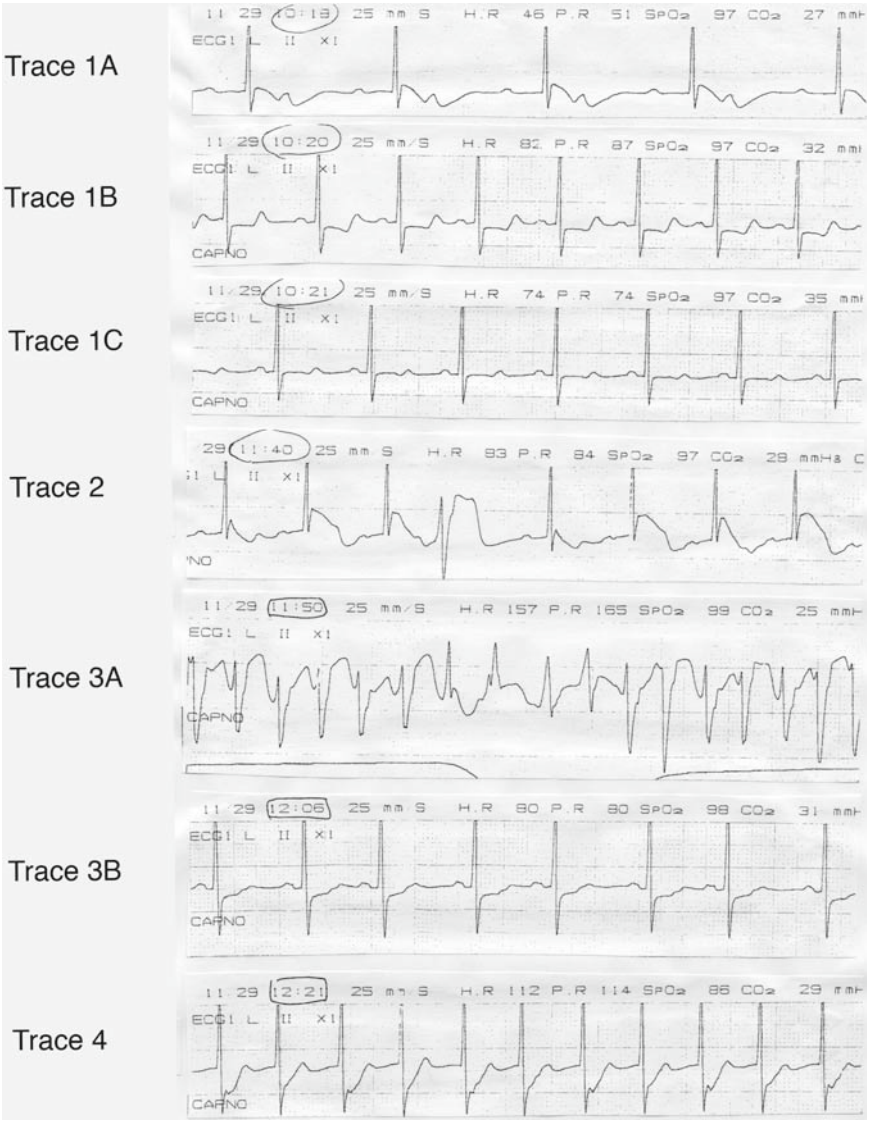

Fig. 1. Electrocardiographic recordings of repeated intraoperative ST changes and ventricular arrhythmia. Time of the recording and the heart rate (HR) of each trace are shown in the *upper left corner of each trace*. *Traces 1A through 1C*. First episode of electrocardiographic (ECG) change (trace 1A); subsequent ST depression and T wave inversion (trace 1B) spontaneously normalized (trace 1C). *Trace 2*. Second episode of ECG change. ST elevation and ventricular premature contractions were successfully treated with intravenous lidocaine. *Traces 3A and 3B*. The third episode of ECG change. Ventricular tachycardia, which necessitated DC defibrillation, temporary chest compression, and epinephrine administration, was recorded in lead II at 10 min after the second episode (*trace 3A*). Only slight ST depression was noted in the ECG after treatment (*trace 3B*). *Trace 4*. The fourth episode of ECG change. Junctional rhythm, ST depression, and severe hypotension, which required external chest compression and intravenous epinephrine administration, were noted 30 min after the episode of coronary spasm

second event, the prostaglandin administration was terminated. At this time, the HR was between 80 and 90 bpm, and the systolic blood pressure was between 90 and 110 mmHg. The surgical procedure proceeded after consultation with the neurosurgeon, as the second episode of ECG change had been successfully treated with lidocaine. The rest of the anesthetic regimen remained constant during these periods, and arterial blood gas analysis revealed no abnormalities (pH, 7.45; Pa_{CO_2}, 38 mmHg; Pa_{O_2}, 170 mmHg; hemoglobin [Hb], 13.1 $g \cdot dl^{-1}$). Ten minutes after the second episode, significant ST elevation and pulseless ventricular tachycardia was noted (Fig. 1, trace 3A). This life-threatening arrhythmia did not respond to 100 mg intravenous lidocaine and was immediately treated with DC defibrillation and 1 mg of intravenous epinephrine. The ECG returned to sinus rhythm with a rate of 90 bpm and moderate ST depression. The systolic blood pressure was stabilized around 120 mmHg after transient hypertension due to intravenous epinephrine administration (Fig. 1, trace 3B). After this event, N_2O was terminated but propofol was continued at the same rate as previously. Cardiovascular support and vasospasm prophylaxis consisted of a continuous infusion of dopamine, nicardipine, lidocaine, and diltiazem, and the surgery was postponed due to these adverse cardiovascular conditions. Multiple episodes of hypotension with systolic blood pressure around 80 mmHg occurred during dural and cranial closure and these were treated with intravenous ephedrine and phenylephrine. Thirty minutes after the third event, clinical cardiac arrest following severe hypotension (systolic blood pressure below 60 mmHg) occurred and was successfully treated with 1.5 min of chest compression and repeated epinephrine administration. ECG monitoring at this time revealed an AV junctional rhythm with a rate of 112 bpm and marked

ST depression (Fig. 1, trace 4). High-dose continuous epinephrine ($0.5\ \mu g \cdot kg^{-1} \cdot min^{-1}$) administration was added to the medications, and stable hemodynamics was achieved during craniotomy closure. At the end of the surgery, these intraoperative events were diagnosed as coronary vasospasm by a consulting cardiologist. Subsequent echocardiographic study demonstrated no pathologic lesion, no signs of inadequate preload, and well-preserved ventricular contractility. Postoperative chest radiograph and blood gas analysis revealed no abnormal findings, and the patient was transferred to the neurological intensive care unit (ICU) and mechanically ventilated. On arrival in the ICU, the HR was 86 with a sinus rhythm, and the systolic blood pressure was between 120 and 140 mmHg with dopamine infusion at a rate of $3\ \mu g \cdot kg^{-1} \cdot min^{-1}$. Neurologically, the patient showed dilated pupils with a sluggish light reflex at the end of surgery, but she had regained consciousness 60 min later without any neurological signs or symptoms. The patient was extubated after the administration of epinephrine was terminated. During and after this recovery period, no ECG abnormality was found, and the patient was discharged without invasive diagnostic testing.

Discussion

The three episodes of ST segment elevation and ventricular arrhythmia described in this report were most likely caused by coronary spasm, because the patient had no signs of preoperative myocardial ischemia and no apparent imbalance of the myocardial oxygen demand-and-supply relationship at the time of these events [3]. Based on our MEDLINE literature search, 17 relevant case reports written in English about intraoperative coronary spasm during noncardiac surgery were located. Additionally, 21 case reports with English abstracts were found in the Japanese literature. The case we have described is characterized by the fact that the multiple events of coronary spasm took place during a single anesthesia course. From this perspective, we believe this report may provide some additional information about the interaction between coronary spasm and anesthesia. Although the pathophysiology of coronary spasm remains to be elucidated, involvement of endothelial dysfunction and the autonomic nervous system is suggested [4]. In the context of anesthetic management, sympathetic activation due to inadequate depth of anesthesia, parasympathetic activation due to vagal stimulation, neostigmine and neuraxial blockade, alkalosis, and hypotension have been implicated as triggers of coronary spasm [2,4,5]. We believe that the HR and blood pressure in our patient precluded inadequate anesthetic depth or myocardial hypoperfusion at the time of the coronary spasm. At this time, the blood gas analysis revealed no hypocapnia or alkalosis. It is not readily known whether all episodes were triggered by the same mechanism, however, three possible causes may have been involved. First, the administration of propranolol and prostaglandin E_1 may have triggered a coronary spasm. Several reports implicate propranolol as a triggering agent of coronary spasm, by blocking sympathetic activity and causing parasympathetic dominance [6–8]. Although the majority of investigations have revealed a protective effect of β-blockade on myocardial ischemia [9], β-blockade may cause spastic vasoconstriction under certain conditions. Whether β-blockade may trigger coronary spasm or protect the post-ischemic myocardium warrants further investigation. Prostaglandin E_1 is generally regarded to have a myocardial protective effect [10]. However, several anecdotal reports in the Japanese literature have demonstrated a temporal coincidence between prostaglandin E_1 administration and the occurrence of coronary spasm [11–13]. In our patient, the fact that ECG change relevant to coronary spasm occurred only during prostaglandin E_1 infusion may suggest this possibility. Second, stimulation of the vagal nerve during neurosurgical manipulation may be involved as an underlying mechanism of coronary spasm [12]. As each episode in our patient occurred during craniotomy and during surgical exposure of the cerebral aneurysm, it is possible that the vagal nerve may have been stimulated at the time of each episode. Third, propofol-based anesthesia may have contributed to the coronary spasm. One laboratory investigation demonstrated that propofol was less protective against coronary spasm than sevoflurane [14]. Of the 115 cases of coronary spasm reported during the period from 1968 to 1998, 32% of the patients were anesthetized with an inhalational agent, while 11% were anesthetized with an intravenous agent [2]. However, the contribution of anesthetic choice to the occurrence of coronary spasm is not readily understood, because the total number of cases is not known.

In summary, we have reported a case of recurrent episodes of ST elevation and ventricular tachycardia during neurosurgical anesthesia. Coronary spasm is most likely implicated as an underlying mechanism of these symptoms. Although the precise mechanisms remain unclear, multiple factors, such as β-blockade, prostaglandin E_1 administration for deliberate hypotension, vagal stimulation elicited by surgical manipulation, and propofol may have been involved. This case reminds us that even transient ST elevation and a few ventricular premature contractions that are spontaneously alleviated may be a sign of more clinically significant coronary spasm. Meticulous attention is needed to circumvent possible triggering conditions and to provide definitive prophylaxis after these episodes.

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