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CLINICAL REPORT

A case of intraoperative coronary artery spasm in a patient with vascular disease

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Abstract A 66-year-old man with a history of longtime smoking, untreated hypertension, hyperlipidemia, and impaired glucose tolerance but no history of myocardial infarction or angina pectoris was scheduled for right aortofemoral bypass and thromboembolectomy for arteriosclerosis obliterans with right common iliac and right popliteal arterial thrombus. Epidural anesthesia and general anesthesia were administered without obvious ECG changes. Just after skin incision, ST elevation in leads II and V5 and a short run of ventricular tachycardia with frequent premature ventricular contractions (PVCs) were recorded on the ECG monitor, and the patient's blood pressure suddenly decreased within a few seconds. On noticing these changes, we suspected coronary artery spasm (CAS) and rapidly administered vasodilators and vasopressors to stabilize hemodynamics and ECG changes. Transesophageal echocardiography (TEE) showed basal to mid- and anteroseptal to inferior wall motion hypokinesis that gradually returned to normal during observation. Even in patients without coronary disease but with systemic arteriosclerosis, it is important to consider the possibility of perioperative CAS and not to overlook ECG changes. Immediate diagnosis and treatment are essential.

Keywords Coronary artery spasm · Vascular disease · General and epidural anesthesia

Introduction

Coronary artery spasm (CAS) can cause angina and myocardial ischemia, regardless of the presence of fixed coronary artery disease. CAS is a potential and not so rare complication that can occur during general and epidural anesthesia [1]. Thus, it is very important to remain current as to the causes, diagnosis, and treatment of this potentially serious condition. Here, we present a unique case of intraoperative CAS in a patient with peripheral vascular disease. Immediate diagnosis of CAS by ECG monitoring and transesophageal echocardiography (TEE) and treatment by both vasopressors and vasodilators were effective in treating the CAS and in preventing myocardial ischemia and future cardiac events in this patient.

Case report

A 66-year-old man (height, 155 cm; weight, 51.5 kg) was diagnosed as having arteriosclerosis obliterans with right common iliac and right popliteal arterial thrombus and was treated conservatively with continuous intravenous infusion of heparin and prostaglandin E_1. However, ischemic leg pain, limping, and coldness in his distal right leg became worse, and he was scheduled for right aortofemoral bypass and thromboembolectomy.

The patient had a history of longtime smoking (Brinkmann index = 1,000), untreated hypertension and hyperlipidemia,

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and impaired glucose tolerance (HbA1c, 6.1%). He had no history of myocardial infarction or angina pectoris and no limitations on physical activity. Preoperative laboratory data were as follows: hemoglobin, 14.8 g/dl; hematocrit, 42.5%; total creatine phosphokinase (CPK), 490 IU/l (reference range, 30–110 IU/l); CK-MB, 36 IU/l (reference range, 25.5–55 IU/l); ALT (alanine aminotransferase), 67 IU/l (reference range, 4–37 IU/l); blood urea nitrogen (BUN), 18.6 mg/dl (reference range, 10–25 mg/dl); and creatinine, 1.13 mg/dl (reference range, 0.6–1.15 mg/dl). Preoperative 12-lead ECG and transthoracic echocardiography were normal. He did not undergo preoperative coronary angiography, coronary computed tomography (CT) angiography, or stress testing.

No preoperative medication was given. Continuous intravenous infusion of heparin was stopped 12 h before surgery. Routine monitoring [ECG leads II and V5, non-invasive blood pressure (BP) measurement, pulse oximetry] was begun on patient arrival in the operating room. The ECG showed normal sinus rhythm at a rate of 72 beats/min (bpm), and his BP was 150/86 mmHg. An 18-gauge epidural catheter was inserted into the thoracic 11/12 (T11–T12) interspace and was threaded 6 cm into the epidural space. After a negative aspiration test, 2.5 ml 1% lidocaine without epinephrine was injected with no change in heart rate (HR) and sensory block, and 8 ml 0.2% ropivacaine was injected. General anesthesia was induced with intravenous propofol 60 mg, fentanyl 0.1 mg, vecuronium 8 mg, and sevoflurane 2–4%. After muscle relaxation was achieved, an 8.5-mm endotracheal tube was easily placed to a depth of 21 cm. Immediately thereafter, HR and BP temporarily increased to 88 bpm and 172/92 mmHg, respectively, and gradually decreased to 60 bpm and 140/64 mmHg. General anesthesia was maintained with 33% nitrous oxide and 1% sevoflurane in oxygen. The patient was mechanically ventilated with a tidal volume of 450 ml and respiratory rate of 8 breaths/min to maintain $PaCO_2$ at 35–40 mmHg under end-tidal CO_2 monitoring. Arterial BP was continuously monitored via a left radial artery catheter.

Eight minutes after tracheal intubation, arterial BP gradually decreased from 134/60 to 92/52 mmHg, and HR gradually increased from 62 to 74 bpm. Ephedrine 6 mg was given intravenously. Arterial BP increased to 118/58 mmHg, and HR increased to 82 bpm without ECG change. The operation started 35 min after first administration of ephedrine 6 mg, and just before skin incision, the patient's BP was 112/54 mmHg and his HR was 78 bpm.

Just after skin incision, ST elevation and a short run of ventricular tachycardia with frequent premature ventricular contractions (PVCs) were recorded on the ECG monitor (Fig. 1). The patient's BP was 126/62 mmHg and HR was 82 bpm. We suspected CAS and decided to administer

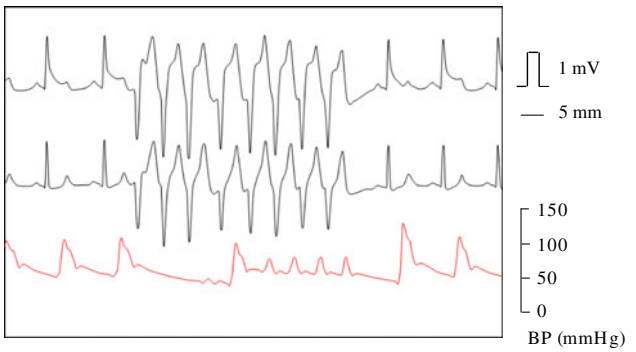

Fig. 1 From the top, ECG leads II and V5 and the arterial blood pressure waveform are presented. ST elevation and a short run of ventricular tachycardia with frequent premature ventricular contractions (PVCs) were recorded by the monitor

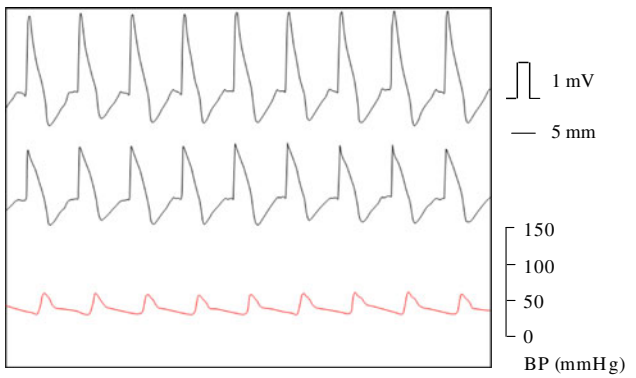

Fig. 2 Tracings are presented as in Fig. 1. The ECG waveforms showed ST elevation

isosorbide dinitrate (ISDN) and perform TEE examination to understand the reason for the unstable hemodynamics.

Within 30 s after skin incision, the patient's BP suddenly decreased from 122/64 to 70/38 mmHg. HR was 88 bpm with what appeared to be either massive ST elevation or wide-QRS tachycardia displayed on monitored leads II and V5 (Fig. 2). Immediately after recognizing this, we administered ISDN 0.5 mg twice (1.0 mg total) for the treatment of massive ST elevation caused by suspected CAS. We then administered ephedrine 6 mg twice (12 mg total) to counter unstable hemodynamics, but the BP remained low. We administered additional ephedrine 6 mg and lidocaine 60 mg to treat for the possibility of wide-QRS tachycardia. However, ECG changes and hemodynamics remained unstable. About 2.5 min after skin incision, BP remained at 68/38 mmHg, and HR was 92 bpm. We administered an additional 1.0 mg ISDN and noradrenaline 10 μg twice (20 μg total), and ECG changes gradually resolved about 3 min after skin incision (Fig. 3). However, the BP still remained low (86/44 mmHg), and HR was 88 bpm. To improve hemodynamics and coronary perfusion, we administered noradrenaline 5 μg, ISDN

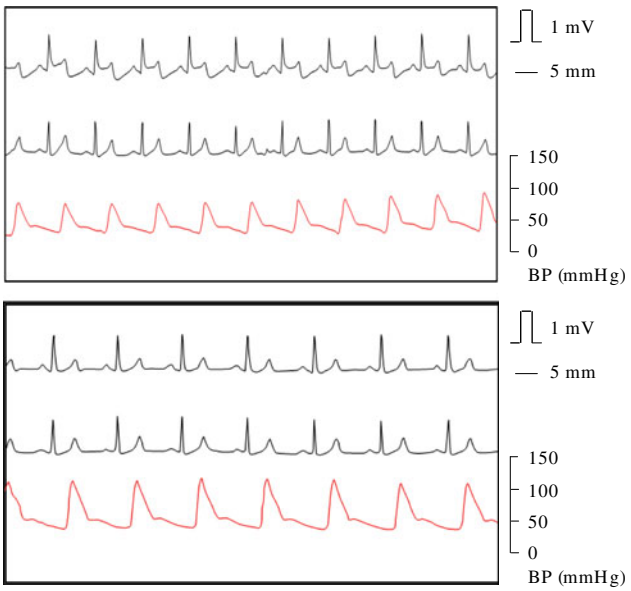

Fig. 3 Tracings are presented as in Fig. 1 in *both panels*. ECG changes improved within a few minutes of treatment

0.5 mg, and additional lidocaine 30 mg for arrhythmia prevention. At 4 min after skin incision, the patient's BP had risen to 108/52 mmHg, and HR was 82 bpm.

About 5 min after skin incision, ECG change and hemodynamics had almost stabilized (Fig. 3). The BP was 114/58 mmHg, and HR was 66 bpm. We performed TEE, which showed basal to mid- and anteroseptal to inferior wall motion hypokinesis that gradually returned to normal during observation. We strongly suspected participation of CAS from the changes on the monitor ECG and TEE findings. So, we began infusions of ISDN (0.5 μg/kg/min) and diltiazem (1 μg/kg/min) to treat and prevent further spasm. Continuous dopamine infusion (2.0 μg/kg/min) was also started to support circulation and BP.

After stabilizing the patient, we concluded that aortofemoral bypass would be too stressful for him and performed femoro-femoral bypass and thromboembolectomy instead. No further ECG changes were observed during surgery. The patient was extubated in the operating room without complications such as ischemic chest pain and was transferred to the cardiac care unit. Postoperative CPK was 550 IU/l, CK-MB was 44 IU/l, and troponin T was negative. No evidence of myocardial ischemia was seen on 12-lead ECG or by transthoracic echocardiography, strongly suggesting that little ischemic change had occurred, thanks to the immediate diagnosis and treatment of the CAS.

Coronary angiography performed 2 weeks postoperatively showed no organic stenosis in any coronary artery. Following intraluminal administration of acetylcholine, however, spasm occurred in both the right and left coronary arteries (Fig. 4). The coronary arteries returned to their normal size after administration of intracoronary nitroglycerin. The patient suffered no further cardiac attacks during his hospital stay. He was prescribed nifedipine (40 mg/day) for the prevention of CAS and treatment of hypertension and has been followed without symptoms of CAS by a cardiologist. Consent was obtained from the patient to publish this case report.

Discussion

Coronary artery spasm, the result of abnormal contraction of coronary vascular smooth muscles, is involved in various coronary arterial diseases, such as vasospastic angina, myocardial infarction, and sudden death [1]. Chang and Hanaoka [2] reported in detail on CAS during surgery. Mean patient age was 58 ± 13 years. The majority of patients were men (75%), were Japanese (78%), and had no history of chest pain (75%). The prognosis of CAS during anesthesia was relatively good. Regional anesthesia, vasopressors, hyperventilation, hypotension, and inadequate depth of anesthesia were noted as major contributing factors. Sidi et al. [1] also reviewed CAS in detail and found results very similar to those of Chang et al. In addition to the traditional contributing factors already mentioned, oculocardiac reflex [3], anaphylaxis [4, 5], carotid sinus stimulation [6], neurological procedures [7–9], rocuronium [10], dolasetron [11], prostaglandin E_1, and beta-adrenergic blockade [12] are also recently reported factors.

In the present patient, systemic arteriosclerosis was likely caused by smoking, hyperlipidemia, and mildly impaired glucose tolerance. These arteriosclerotic changes might also occur in the coronary arteries. Even in the absence of fixed coronary artery disease, endothelial dysfunction resulting in acceleration of coronary artery constriction or disabling of coronary dilation can occur in arteriosclerotic coronary arteries [13]. For the patient with such coronary artery instability, common occurrences during anesthesia such as low BP caused by anesthetic induction, vasopressor use, surgical stress of the skin incision caused by inadequate depth of anesthesia, or relative parasympathetic hyperactivity caused by epidural anesthesia might cause coronary artery hyperconstriction. However, CAS is uncommon. It is not clear whether these are causes of or are coincidental to CAS, and it can sometimes be difficult to explain why such common events might cause CAS in certain instances. Further study is necessary to establish existence of a genetic or environmental component of CAS during anesthesia.

Regional anesthesia such as spinal and epidural anesthesia is associated with cardiovascular complications, most notably hypotension and bradycardia, which often necessitate the use of sympathomimetic drugs such as ephedrine and

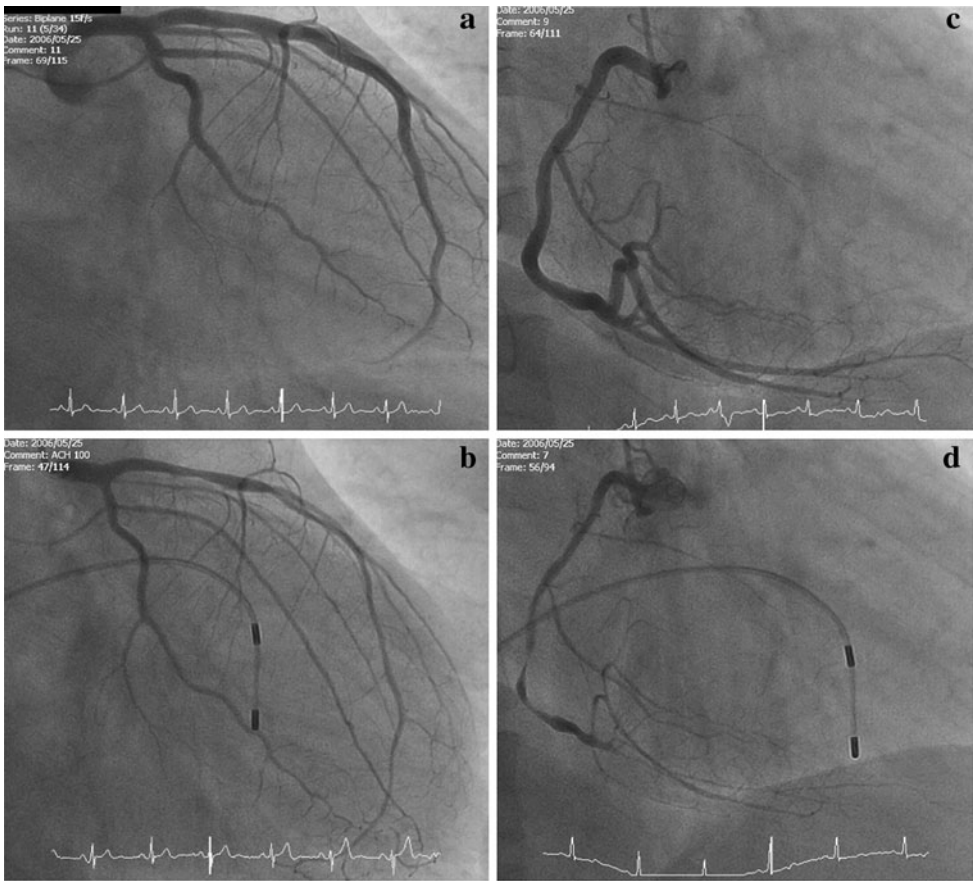

Fig. 4 Coronary angiography after surgery. No organic stenosis was observed in the resting state in either the left or right coronary arteries (**a**, **c**). However, intraluminal administration of acetylcholine produced spasm of both the left and right coronary arteries (**b**, **d**)

phenylephrine. In the present patient, anesthetic induction brought about transient low BP, which we initially treated with ephedrine. Several case reports describe ephedrine use as the cause of CAS [1, 2, 14–16]; thus, treating hypotension and bradycardia caused by regional anesthesia with these drugs might cause CAS. However, the ECG in our patient did not show ST-T abnormality until approximately 35 min after first ephedrine administration. Although we cannot deny the potential possibility that CAS at the time of skin incision was a result of earlier ephedrine administration, we did not believe this to be the direct cause. Sympathetic excitation above the level of partial or complete sympathetic blockade is also thought to cause CAS associated with epidural anesthesia [17]. It is possible that increased sympathetic activity above the level of sympathetic block rather than complete sympathetic blockade could be related to the onset of CAS in our patient. Our epidural block may not have been adequate because the patient experienced postoperative pain. With potentially ineffective epidural analgesia, the stress of the skin incision might have been enough to cause CAS in our patient, even though he was anesthetized by 33% nitrous oxide and 1% sevoflurane.

Diagnosis of CAS was ultimately made on the basis of ECG changes in monitored leads II and V5 during surgery. It is impossible for the anesthetized patient to complain of chest pain, and it is usually difficult to stop a surgical procedure to transfer the patient to the cath lab for coronary angiography to verify the presence of CAS, in contrast to the case reported by Ornek et al. [18]. TEE is useful to image new regional wall motion abnormalities during general anesthesia. At the time, we could not differentiate with certainty whether the ECG changes occurring in our patient were those of ST elevation or a wide QRS. TEE showed new regional wall motion abnormalities even after stabilization of the ECG changes and hemodynamics. TEE revealed inferior wall hypokinesis, which disappeared within a few minutes. This finding enabled us to start continuous infusion of vasodilators.

Chang and Hanaoka [2] noted that more than half their patients during surgery experienced severe hypotension, and 30% developed cardiovascular collapse as a result of CAS. Sidi et al. [1] suggested that CAS can lead to bradycardia, complete atrioventricular (AV) block, ventricular tachycardia, and, eventually, asystole. Thus, appropriate and timely treatment must be provided to prevent myocardial ischemia when ST changes appear.

We administered coronary vasodilators and vasopressors simultaneously in our patient. A number of existing reports

address use of vasopressors for the treatment of unstable hemodynamics resulting from CAS [1, 2, 4, 5, 9, 15–20]. We thought it necessary to maintain coronary perfusion pressure with vasopressors to counteract the reduced coronary circulation caused by CAS. However, vasopressors might not only produce CAS but also increase cardiac muscle oxygen consumption. Fortunately, these treatments resulted in immediate recovery and hemodynamic stabilization without worsening CAS, allowing our patient to recover without myocardial ischemia. However, once CAS occurs, immediate administration of a full dose of coronary dilators is recommended [1, 2, 21].

In this patient experiencing CAS during vascular surgery, immediate diagnosis on the basis of changes in monitored ECG leads and TEE findings and appropriately timed administration of both coronary vasodilators and vasopressors were effective in the treatment of CAS. The possibility of perioperative CAS in the patient with systemic arteriosclerosis must always be considered, and ECG changes should not be overlooked. Immediate diagnosis and treatment allowed recovery of our patient without myocardial ischemia.

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