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CLINICAL REPORT

Angiographic documentation of coronary artery spasm induced by anesthesia

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Abstract Many cases of coronary artery spasm during regional or general anesthesia have been reported. To our knowledge, for the first time we report a case of coronary artery spasm induced by general anesthesia that was documented by immediate angiography and treated with intracoronary nitroglycerine. A 68-year-old female was to undergo elective cholecystectomy. General anesthesia was induced by propofol 140 mg, fentanyl 50 μg, and vecuronium bromide 7 mg. Immediately after intubation sudden hypotension (60/40 mmHg) and marked ST-segment elevation on the monitor were noticed. A 12-lead electrocardiogram demonstrated marked ST-segment elevations in inferior leads with reciprocal changes. An immediate diagnostic coronary angiography showed diffuse severe narrowing of the right coronary artery with a non-critical plaque in the midportion. After intracoronary administration of nitroglycerine 0.2 mg into the right coronary artery, there was marked diffuse vasodilatation. A diagnosis of variant angina was made and 2 days of hospital course was uneventful. The patient was discharged taking diltiazem 120 mg two times daily, aspirin 100 mg daily and isosorbidemononitrate 60 mg daily.

Keywords Coronary · Spasm · Variant · Angina · Angiography

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Introduction

Coronary artery spasm results in myocardial ischemia that may lead to angina, arrhythmia, and myocardial infarction [1]. Many cases of coronary artery spasm during regional or general anesthesia have been reported [2]. In those cases the diagnosis was suggested by a history of angina at rest with typical electrocardiogram changes or with a coronary angiography showing coronary spasm that was provoked by ergonovine or acetylcholine. To our knowledge, for the first time we report a case of coronary artery spasm induced by general anesthesia that was documented by immediate angiography and treated with intracoronary nitroglycerine.

Case report

A 68-year-old female (ASA physical status II) was to undergo elective cholecystectomy. She had a history of hypertension that was well controlled with losartan 50 mg plus thiazid 12.5 mg daily. There were no other risk factors for coronary artery disease. Physical examination was unremarkable. Blood pressure was 125/85 mmHg. Preoperative resting ECG showed normal sinus rhythm with a rate of 72 bpm and no abnormalities. Chest radiography and laboratory data revealed no abnormalities. The patient arrived at the operating room without premedication and intravenous access was established. General anesthesia was induced by propofol 140 mg, fentanyl 50 μg, and vecuronium bromide 7 mg. The trachea was intubated without difficulty. Immediately after intubation sudden hypotension (60/40 mmHg) and marked ST-segment elevation on the monitor were noticed. Heart rate was 67 bpm. A 12-lead electrocardiogram demonstrated marked ST-segment elevations in inferior leads with

reciprocal changes (Fig. 1). The surgery was cancelled. Normal saline was administered intravenously, beginning with a bolus of 100 ml followed by 50 ml increments every 5 min. A diagnosis of suspected acute inferior myocardial infarction was made by a consultant cardiologist. In order to perform primary percutaneous coronary intervention, the patient was transferred to the catheterization laboratory in 10 min. An immediate diagnostic coronary angiography showed diffuse severe narrowing of the right coronary artery with a non-critical plaque in the midportion (Fig. 2). The left coronary artery was normal. After intracoronary administration of nitroglycerine 0.2 mg into the right coronary artery, there was marked diffuse vasodilatation (Fig. 3). Blood pressure stabilized at 100/60 mmHg. Nitroglycerine infusion 10 μg/min was started. ST-segment elevation began to decline and an electrocardiogram showed no ischemic changes 10 h later. Cardiac enzyme measurements were within normal limits (CK-MB 8 IU/L normal range 0–16 IU/L, troponin I 0.01 ng/ml normal range 0–0.1 ng/ml), thus ruling out myocardial infarction. A diagnosis of variant angina was made and 2 days of hospital course was uneventful. The patient was discharged taking diltiazem 120 mg two times daily, aspirin 100 mg daily and isosorbidemononitrate 60 mg daily.

Discussion

Coronary artery spasm during regional or general anaesthesia has rarely been reported [2]. It has been implicated as a cause of syncope, ST-segment elevation, atrioventricular block, asystole, ventricular tachyarrhythmias, myocardial infarction, and death [1].

Coronary artery spasm is established as a cause of Prinzmetal variant angina. These patients do not exhibit classic coronary risk factors, with the exception of smoking, and tend to be younger than patients with typical angina pectoris. Anginal episodes occur generally at rest and associated with ST-segment elevation on ECG. Nitrates are effective in relieving coronary vasospasm and anginal pain [3].

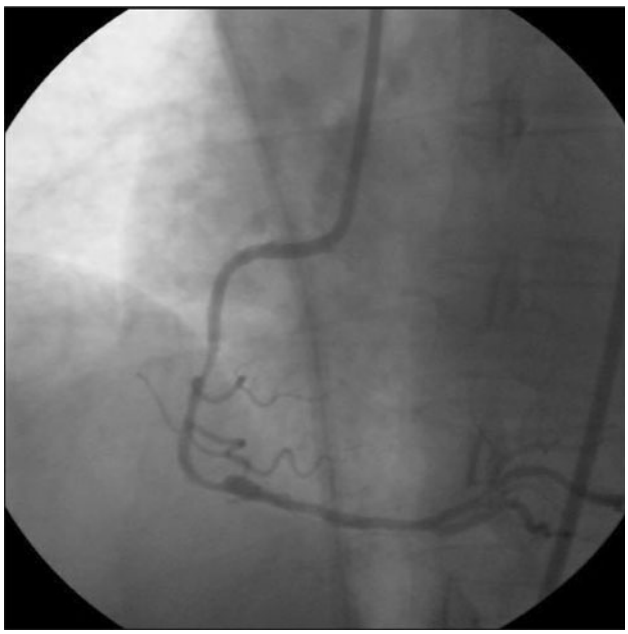

Fig. 2 Coronary angiography showed severe diffuse narrowing of the right coronary artery

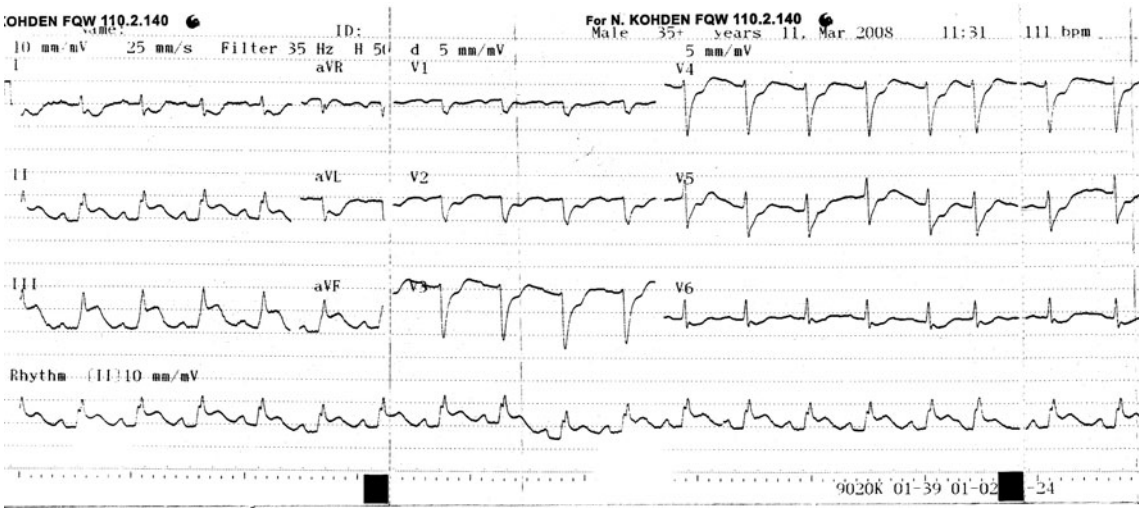

Fig. 1 An electrocardiogram in the operation room demonstrated marked ST-segmental elevations in inferior leads and reciprocal ST-segment depression in the precordial leads

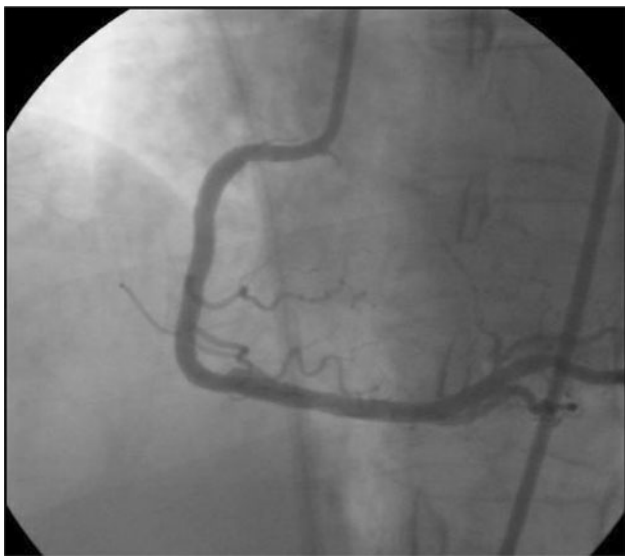

Fig. 3 After intracoronary nitroglycerine administration there was marked diffuse vasodilatation of the right coronary artery

Although coronary angiography frequently demonstrates normal coronary arteries, it is possible that the coronary arteries that are involved in spasm may be in various stages of atherosclerosis, with absent or dysfunctional endothelium [4]. When stenosis is seen with ergonovine or acetylcholine induction, the right coronary artery is usually involved [5]. Coronary spasm may be related to excess alpha adrenergic activity, stimulation of the parasympathetic nervous system, altered humoral factors, increases in blood pH, and imbalance of vasoconstrictor-vasodilator forces [6].

In our patient, coronary artery spasm occurred a few minutes after the administration of propofol plus fentanyl and endotracheal intubation. It is possible that intubation or these agents, via activation of autonomic nervous system, provoked coronary artery spasm. The anxiety of the patient or the inadequate state of anesthesia might be responsible for the coronary spasm. Furthermore coronary spasm might be caused either by allergic reaction to these drugs or by drug-induced hypotension [7]. In addition, a non-critical plaque in the right coronary artery may have made her more susceptible to vasospasm. It has been reported that patients with hypertension have abnormal endothelium-dependent vascular relaxation [8].

Awareness of this variant of coronary artery spasm is the key to successful outcome. The diagnosis of variant angina is usually suggested by a history of angina at rest with ST-segment elevation on ECG. However early recognition during routine preoperative evaluation is not always possible. In this patient, for the first time, we were able to show coronary artery spasm by immediate coronary angiography without ergonovin or acetylcholine induction. The definitive diagnosis of variant angina was made using simultaneous coronary angiography; treatment was with intracoronary nitroglycerine. In this patient, initial diagnosis before angiography was acute myocardial infarction with hypotension. Nitroglycerine should not be given to patients with myocardial infarction and hypotension. In such cases, nitrate-induced venodilation could impair cardiac output and reduce coronary flow, thus worsening myocardial oxygenation rather than improving it. To put the patient in reverse Trendelenburg position, saline infusion and atropine may be helpful. However atropine may be unsafe in cases of variant angina because of coronary spasm. If hypotension persists despite correction of hypovolemia, positive inotropic agents may be indicated. In patients with cutaneous vasoconstriction, therapy with dobutamine is preferable. In cases of hypotension with clinical evidence of vasodilation, phenylephrine hydrochloride may be useful. However this agent should be used with caution because it increases coronary and peripheral vascular tone. In patients with myocardial infarction, if the heart rate is extremely slow (<40 bpm) and associated with hypotension, intravenous atropine can be administered. In cases of persistent bradycardia despite atropine (with a total dose of 2 mg) cardiac pacing is advisable.

Recommended treatments of variant angina are nitrates and calcium-channel blockers. We used intracoronary nitroglycerine in this patient and coronary artery spasm resolved dramatically.

In conclusion, we describe a case of coronary artery spasm during induction of general anesthesia documented and treated using immediate angiography. It is a possible complication of anesthesia and responds to treatment with nitrates and calcium-channel antagonists.

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