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Case Report Open Access

Quick decision and right management in coronary artery vasospasm following on-pump coronary artery bypass grafting

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ABSTRACT

The importance of early diagnosing of coronary artery vasospasm (CAV) which can cause serious, life-threatening hypotension immediately after coronary artery bypass grafting (CABG) is well-known. High endogenous catecholamine levels, vasopressor treatments, vascular endothelial damage due to physical manipulation of the perioperative coronary artery, hypothermia and increased inflammatory response during cardiopulmonary bypass can be counted among the various factors thought to induce CAV in the postoperative period. However, quick decision and right management can be life-saving in CAV. Herein, we present successful treatment of CAV after CABG operation in a 48-year-old active smoker, female patient with cardiological follow-up due to stable angina preoperatively.

Keywords: Angina pectoris; coronary artery bypass grafting, coronary artery vasospasm, myocardial infarction, variant angina.

It was the first time in 1959 that Prinzmetal et al.[1] identified a syndrome with ischemic chest pain which was not stimulated by emotional stress and physical exercise accompanied by myocardial ischemia findings and ST segment elevation. This syndrome, also called variant angina pectoris, was associated with acute myocardial infarction, ventricular tachycardia, ventricular fibrillation, and sudden death.

Variant angina pectoris may exist after coronary artery bypass grafting (CABG) and is usually accompanied by generalized vasospastic diseases such as Raynaud's phenomenon and migraine.[1] While the most common involvement is the right coronary artery (RCA), the left anterior descending artery (LAD) or co-involvement are less frequently observed.[2] Coronary artery vasospasm (CAV) developing after CABG may be manifested by refractory angina, malignant arrhythmias, ST segment elevation on electrocardiography (ECG), myocardial infarction, sudden hypotension, bradycardia and myocardial wall motion abnormalities, and sudden cardiac arrest.

The relationship between coronary artery disease (CAD) and CAV is frequently investigated in the literature. However, there are limited case reports related to CAV after CABG. Herein, we present that successful treatment of CAV after CABG operation in a 48-year-old, active smoker, female patient with cardiological follow-up due to stable angina preoperatively.

CASE REPORT

A 48-year-old female patient was admitted to our clinic with stable angina. The patient, who was not found to be suitable for percutaneous coronary intervention by the cardiologist due to long-segment LAD lesion previously, was operated for single-vessel CABG without any problems. Her medical history revealed active smoking (20 pack year), exercise intolerance and effort angina. There was no abnormality in the blood gas and other respiratory criteria to explain tachypnea (24 breaths/min) after extubation. However, while under nitroglycerin infusion therapy (0.25 µg/kg/min; Perlinganit®, ADEKA Pharmaceuticals, Istanbul, Turkey),

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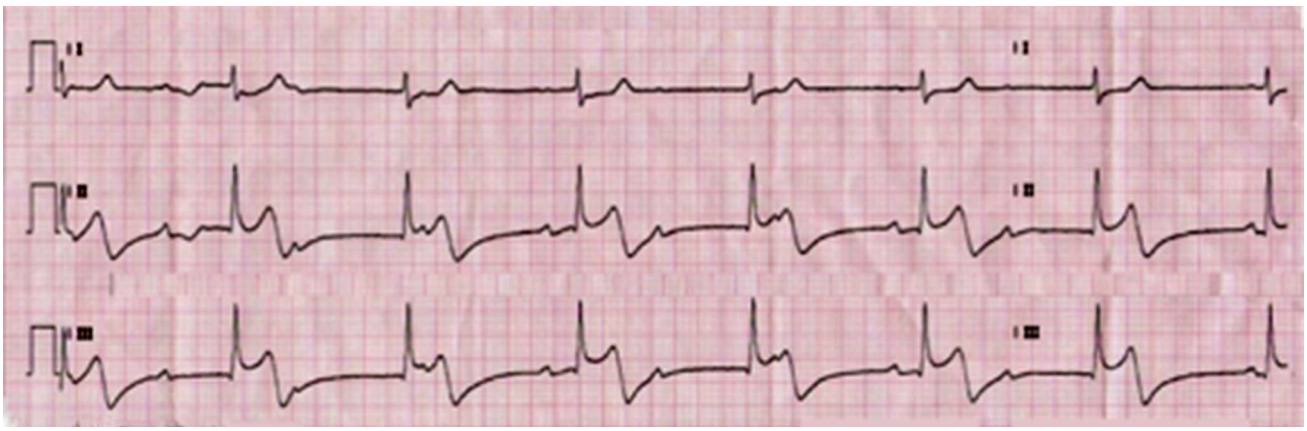

Figure 1. ST segment elevation and third-degree atrioventricular block on electrocardiography.

severe chest pain followed by sudden hypotension (55/35 mmHg), sudden bradycardia (45 bpm), signs of right heart failure symptoms developed at 6 h after the operation. The patient was quickly evaluated by ECG, followed by echocardiography. There were ST segment elevation in the D2-D3 and AvF leads of ECG, and also third-degree atrioventricular block (Figure 1). A pacemaker was inserted through the right internal jugular vein and right ventricular myocardial wall motion abnormality was detected on the echocardiogram. The patient was rapidly taken to coronary angiography after hemodynamic stability was achieved. A written informed consent was obtained from the patient.

Firstly, RCA imaging was performed on coronary angiography. The RCA, which was completely normal preoperatively (Figure 2a), suffered from a totally spasm from the proximal segment to distal segment (Figure 2b). The vessel was reached using a guidewire, and intracoronary nitroglycerin was applied (Figure 2c). However, percutaneous coronary angioplasty (PTCA) was applied to the RCA, as the vasospasm did not regress and hemodynamic instability persisted (Figure 3a). The patient was taken to the intra-aortic balloon pump (IABP) (Datascope CS300®, Getinge, Sweden) support instead of increasing the positive inotropic support due to possible vasospasm effects. The RCA was clear on control angiography, and the patient provided hemodynamic stability rapidly after IABP support. Subsequently, other coronary imaging studies were performed, and the graft anastomosis was well-perfused and clear (Figure 3b). The IABP

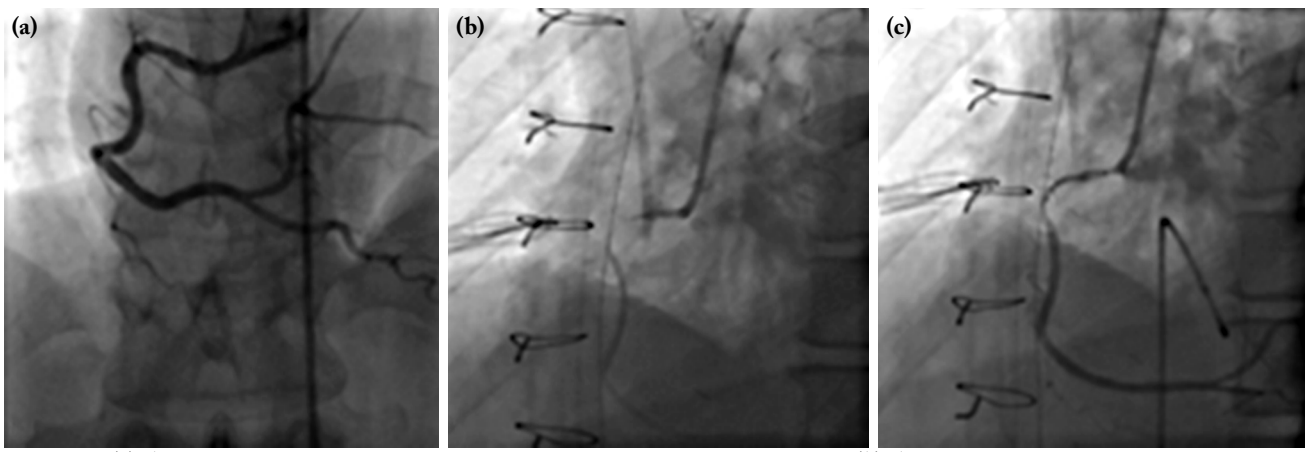

Figure 2. **(a)** Angiographic imaging of right coronary artery before the operation. **(b)** Angiographic imaging of right coronary artery suffering from vasospasm after operation. **(c)** Angiographic imaging of ostial vasospasm continuing after nitroglycerin treatment.

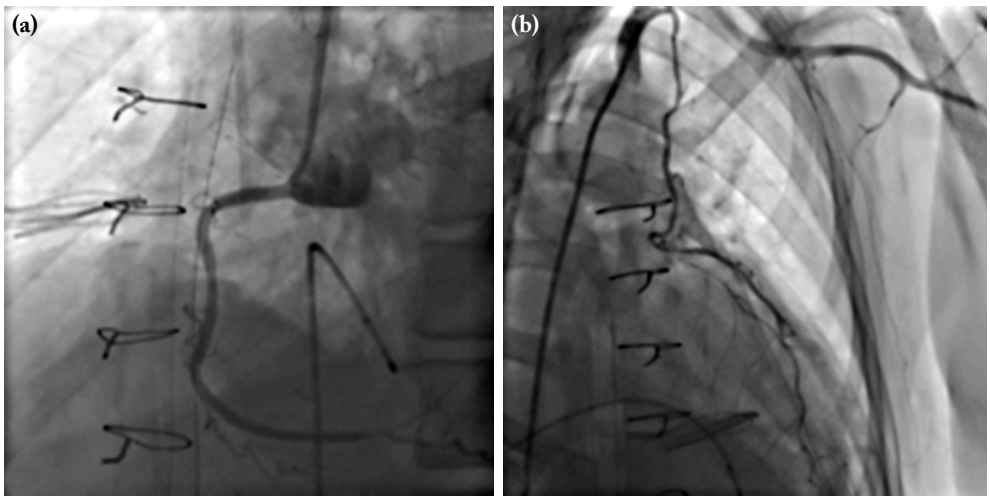

Figure 3. **(a)** Angiographic imaging of right coronary artery after percutaneous coronary intervention. **(b)** Angiographic imaging of left internal mammary artery-left anterior descending artery perfusion after surgery.

support was continued, until the third postoperative day. Meanwhile, intravenous nitroglycerin treatment (0.25-1 μg/kg/min; Perlinganit®, ADEKA Pharmaceuticals, Istanbul, Turkey) with a calcium channel blocker (5-15 mg/h; Diltizem®, Gensenta Pharmaceuticals, Istanbul, Turkey) was applied. On daily echocardiographic evaluation, right ventricular myocardial wall motion abnormality was unable to be seen. The patient was taken to the ward. Nitroglycerin (50 mg/day; Monoket Long®, ADEKA Pharmaceuticals, Istanbul, Turkey) and diltiazem (90 mg/day; Diltizem SR®, Gensenta Pharmaceuticals, Istanbul, Turkey) treatment continued orally. On Day 6 after the operation, the patient was discharged with recommendations.

DISCUSSION

There may be many potential causes of malignant arrhythmia and cardiogenic shock which develop shortly after CABG. Myocardial infarction may also cause this situation after CABG. Despite the technical advances and development of myocardial protection strategies in recent years, perioperative myocardial ischemia associated with increased morbidity and mortality rates is seen in 3.5 to 10% of cases.[3] Myocardial infarction which occurs shortly after CABG may be caused by graft failure or stenosis or incomplete revascularization. However, it should be kept in mind that CAV may cause myocardial infarction and cardiogenic shock, and CAD is a well-known cause of CAV.[4] The latter can be seen at rates ranging from 0.8 to 1.3% after CABG.[4] High endogenous catecholamine levels, the use of vasopressor treatments, vascular endothelial damage due to physical manipulation of the perioperative coronary artery, hypothermia and increased inflammatory response during cardiopulmonary bypass can be counted among the various factors thought to induce CAV in the postoperative period. However, the effects and potency of these factors on patients have not been fully elucidated, yet. In most cases, as in our case, CAV has usually been noted to develop within the first 8 h (mean: 5.6 h) after surgery.[5]

The importance of diagnosing early CAV, which can cause serious, life-threatening hypotension immediately after CABG, has been well-documented in the literature.[4] In our case, emergency coronary angiography was performed to the patient to confirm our suspicion of CAV, to rule out graft failure or possible new-onset stenosis, and to allow intracoronary administration of vasodilators agents, after the patient became hemodynamically suitable for transportation. Calcium channel blockers or nitroglycerin administered intracoronary have been shown to be effective in most cases.[6] However, as in this case report, while nitroglycerin infusion treatment continued, PTCA was applied as the next treatment step to ensure hemodynamic stability in the patient who developed

CAV and the patient was, then, successfully followed with long-term nitrate and calcium channel blocker therapy.

In conclusion, in this case report, we emphasize that the hemodynamic instability developed after a routine and successful operation in the early period following CABG should be managed correctly and that CAV should not be overlooked. In the course of CAV extending to variant angina, myocardial infarction, malignant arrhythmia and even sudden cardiac arrest, we believe that early diagnosis and proper treatment are life-saving for these patients.

Declaration of conflicting interests

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