

# Successful Management of Unremitting Spasm after Myocardial Revascularization Exclusively with Bilateral Internal Thoracic Arteries in Y-graft Configuration: A Case Report

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## ABSTRACT

Coronary artery spasm (CAS) after coronary artery bypass grafting (CABG) is rare, and in time may be fatal for the patient if undiagnosed. The purpose of the present study is to report the case of a patient who survived after experiencing a persistent spasm of all native coronary arteries following successful arterial myocardial revascularization. Furthermore, we aimed to discuss the therapeutic strategies which may prevent the occurrence of a coronary artery spasm in settings of myocardial revascularization, in the context of reviewed specific literature evidences.

## INTRODUCTION

The term coronary artery spasm (CAS) refers to a sudden vasoconstriction of a native coronary artery that causes significant vessel stenosis or even occlusion [Lanza 2011]. CAS has been described in the immediate postoperative period after CABG. CAS has an incidence of 0.8–1.3% [Buxton 1981], and it may occur either during or after CABG, more commonly involving the right coronary artery (RCA). Postoperative CAS should be suspected when abrupt severe hemodynamic deterioration develops after the operation associated with dynamic electrocardiographic (ECG) changes. Specifically, in the presented case, the patient suddenly developed electrical instability with nonsustained ventricular tachycardia (NSVT) and cardiogenic shock (CS). The diagnosis of CAS was confirmed by emergent coronary angiography and should be differentiated from internal mammary coronary artery spasm.

## CASE REPORT

A 74-years-old male patient previously known with angina pectoris, and with percutaneous coronary intervention (PCI)

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drug-eluting stent in the proximal left anterior descending artery (LAD) implanted 1 year ago, was referred to the department of cardiac surgery due to increasing worsening of chest pain. Coronary angiography (CAG) showed a 90% proximal stenosis of the LAD and 50% intrastent stenosis (Figure 1), 50% stenosis on ostial left circumflex artery (LCx), and a spastic RCA (Figures 2 and 3). Preoperative treatment with calcium blockers was started, and that patient was prepared for intervention. The skeletonized left internal thoracic artery (LITA) and right internal thoracic artery (RITA) were harvested through a common median sternotomy. The in situ LITA was anastomosed with the RITA as a free graft in order to form a Y-graft configuration and then normothermic cardiopulmonary bypass (CPB) was started. Bypass grafting to the LAD and to the marginal branch of the LCx was performed by using Y-mammary graft configuration (Figure 4). A bloodless field was maintained using a carbon dioxide blower and the anastomoses were completed using continuous 8-0 Prolene sutures. Moreover, there were no atherosclerotic plaques on the coronary arteries in the anastomosis areas. Total time of cardiopulmonary bypass was 45 minutes; total



Figure 1. Preoperative coronary angiography showing a 90% proximal stenosis of the LAD followed by a 50% intrastent stenosis.

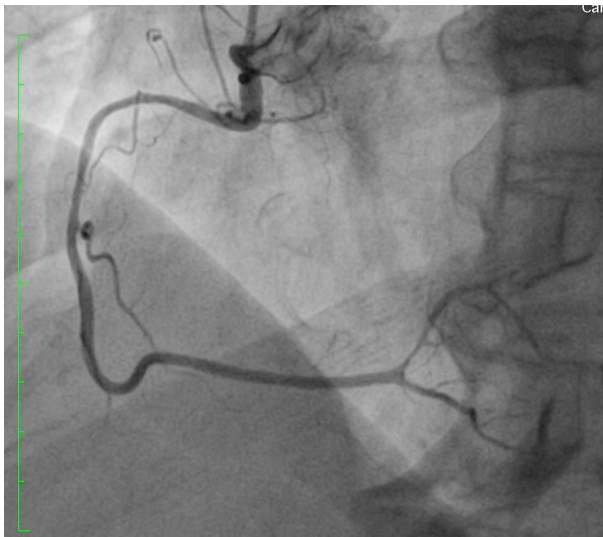


Figure 2. Preoperative coronary angiography showing generalized spasm of the RCA.

aortic clamp time was 23 minutes. After uneventful weaning from the CPB, the patient was transferred to the intensive care unit. At the admission, the patient's blood pressure was between 90-100 mmHg, central venous pressure (CVP) was 5 mmHg, without any inotrope drug but with 100 ng/kg/min norepinephrine. Abrupt severe hemodynamic deterioration developed 2 hours after the operation while the patient was still on mechanical ventilation. ECG showed ST horizontal depression in leads II, III, aVF, and V1-V3, and frequent episodes of NSVT. The ECG signs of inferior wall ischemia were persistent, and systolic arterial blood pressure dropped below 50 mmHg. Vasoactive support was initiated with dobutamine at a maximum of 11.1 mcg/kg/min and norepinephrine dose was raised to 1000 ng/kg/min and adequate



Figure 3. Preoperative coronary angiography showing normal RCA after intracoronary nitroglycerine infusion.

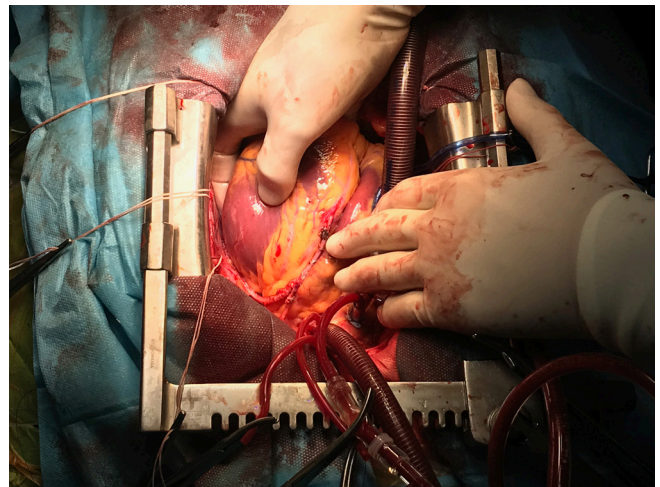


Figure 4. Intraoperative view of the "Y" configuration with the mammary arteries.

hemodynamic parameters were obtained. Values of troponin-I at 2 hours, 6 hours, and 12 hours were 1.59 ng/mL, 1.31 ng/mL, and 1.21 ng/mL, respectively (local laboratory range 0-0.02 ng/mL), and values of creatine-kinase MB (CK-MB) at 2 and 12 hours were 69 UI/L and 121UI/L, respectively (local laboratory range 7-25 UI/L). Emergency transthoracic echocardiography demonstrated a significant decrease of the left ventricular ejection fraction (LVEF) from 50% to 25%, and the patient was immediately transported to the catheterization lab in order to evaluate the patency of the grafts. CAG revealed normal patency of the arterial grafts with severe spasm on all the native coronary arteries (Figure 5). Intracoronary nitroglycerine infusion partially solved the spasm on the LCx, while RCA spasm did not respond to the initial dose

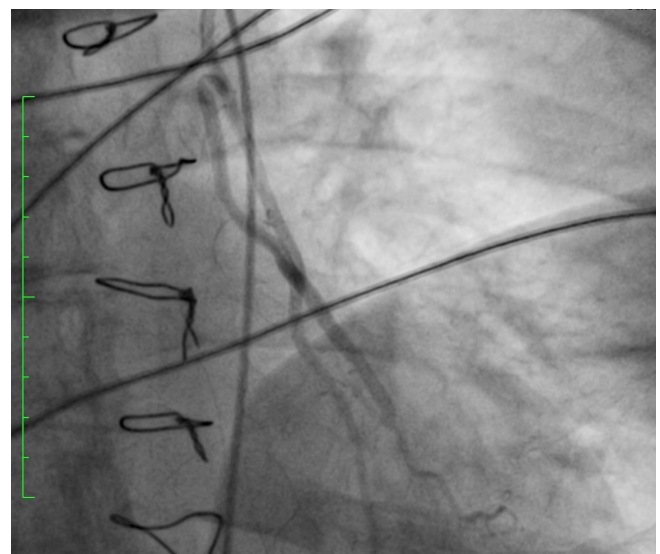


Figure 5. Postoperative coronary angiography showing normal patency of the arterial grafts with severe spasm on the native coronary arteries.

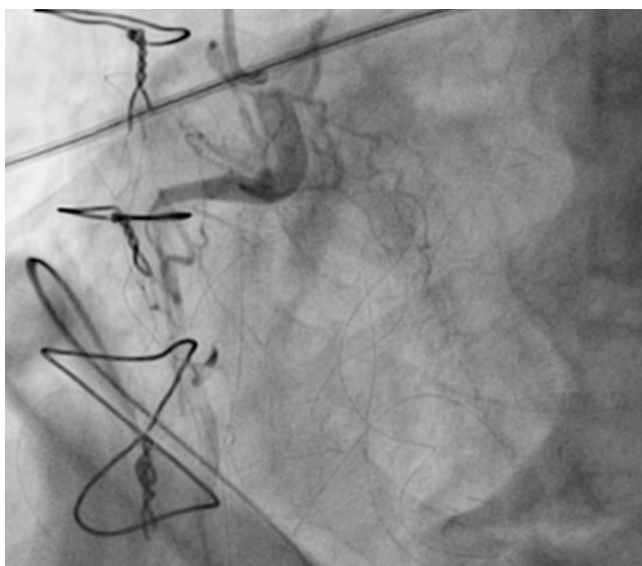


Figure 6. Postoperative coronary angiography showing severe spasm on the RCA after intracoronary nitroglycerine infusion.

of IV nitroglycerine (Figure 6), and attempt of RCA stenting was unsuccessful due to RCA tortuosity. Also, intraaortic balloon pump (IABP) insertion was not possible due to extensive peripheral artery disease. In the intensive care unit, adequate volume replacement therapy, continuous IV infusion of nitroglycerine, and administration of IV calcium channel blockers (diltiazem hydrochloride-initial bolus administration of 20 mL followed by continuous intravenous infusion 1 mg/mL, 10 mL/h, 24 h), concomitant with reducing the doses of vasoactive drugs, helped to slowly improve hemodynamic status. The patient was extubated on the second postoperative day and did not develop any organ dysfunction. The peak CK-MB was 130 U/L on the first postoperative day. The patient was discharged postoperatively on day 9 with diltiazem 40 mg per day administration for 6 months. Echocardiography showed a LVEF of more than 40% on the second day and normalization of the left ventricular function without any ventricular wall motion abnormalities at discharge.

## DISCUSSION

CAS determines narrowing of the coronary arteries due to the contraction of smooth muscle tissue in the walls of the vessels. There are very few data in literature regarding the incidence of postoperative CAS. Recognizing the factors that can lead to this complication is difficult and thus the establishment of a specific treatment is very commonly delayed [Lemmer 1998]. Careful preoperative cardiovascular examinations of the patient's medical records by a cardio team (cardiologist, cardiac surgeon, and anesthesiologist) very often fails to identify a patient's risk of developing arterial spasm. If the risk is suspected, the patient must be operated after administration of vasodilator drugs and postoperative administration of these drugs should be continued. Postoperative

manifestations of coronary arterial spasm mainly depend on the at-risk myocardial territory, and include a diverse range of electrical instability, arrhythmia, ST segment changes and rise of myocardial necrosis markers, which can lead to hemodynamic instability and cardiac arrest. The precise diagnosis of CAS refractory to drug therapy can be established only by urgent coronary angiography [Buxton 1981]. It may occur either during or after CABG, more commonly involves the RCA, and has an incidence of 0.8–1.3% [Buxton 1981]. CAS may be due to surgical manipulation or compression by chest tubes [Marquez 2006], hypomagnesemia, alpha adrenergic stimulation, hypothermia, and damaged platelet release of vasospastic amine thromboxane A<sub>2</sub> [Buxton 1982]. CAG represents the fastest and most accurate diagnostic option for native vessel coronary spasm in patients with previous CABG. When severe CAS is identified, it can be most effectively treated during coronary angiography with intracoronary administration of vasodilators (eg: glyceryl trinitrate, papaverine, verapamil) through native arteries or a bypass graft [Mayumi 1994; Pichard 1980] or extensive angioplasty and stenting of the involved spasm vessel [Schena 2007]. Intravenous nitrates and calcium blockers should be instituted in the immediate postoperative period [He 2016] with adequate mechanical circulatory support. However, in some instances, CAS does not respond to pharmacotherapies, requiring percutaneous cardiopulmonary support [Hiroki 2014]. Note that in patients at risk of spasm, untimely discontinuation of IV vasodilators during the perioperative period may be harmful, even in the presence of patent CABG.

## Conclusion

CAS after arterial myocardial revascularization is very often undetected or even misdiagnosed but, notably, this condition should not be neglected due to the high rate of complications. Although, the first analysis of perioperative coronary angiography may be suggestive of CAS, sometimes CS may be the first manifestation of CAS and only emergent CAG supports the diagnosis. Staged interventions including angiographic procedures, intracoronary vasodilator drugs administration, coronary stenting, percutaneous mechanical circulatory support (IABP, Impella, ECMO), preoperative treatment with diltiazem, adequate volume replacement, and initiation of a continuous IV infusion of nitroglycerine and diltiazem postoperative, concomitant with substantially reducing doses of inotropic drugs, may improve the hemodynamic status of the patient.

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