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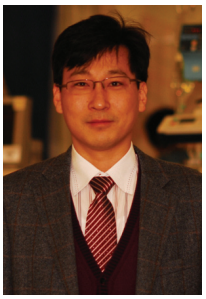
The Role of Percutaneous Cardiopulmonary Support in the Treatment of Native Coronary Spasm after Coronary Artery Bypass Grafting



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ABSTRACT

Postoperative coronary artery spasm following off-pump coronary artery bypass is a rare and unpredictable complication. The clinical manifestations following spasm vary, depending on the severity or the affected vessels. In serious cases, it can proceed to circulatory collapse and cardiac arrest. Coronary angiography with infusion of coronary vasodilators has been a well-established diagnostic and therapeutic tool. We present a patient who was successfully resuscitated with an intra-aortic balloon pump (IABP) and percutaneous cardiopulmonary support (PCPS) as initial stabilization because of an inability to proceed to angiography. Furthermore, we demonstrated the effectiveness of IABP and PCPS for restoring cardiac function.

INTRODUCTION

Native coronary artery spasm is a rare and serious complication after coronary artery bypass grafting [Lemmer 1988; Paterson 1998]. The early diagnosis and prompt treatment of this complication are necessary to prevent its potentially lethal consequences. This report presents angiographic confirmation of native coronary artery spasm following successful off-pump coronary revascularization, which proceeded rapidly to sustained cardiac arrest. Furthermore, we demonstrated the effectiveness of an intra-aortic balloon pump (IABP) and percutaneous cardiopulmonary support (PCPS) for initial stabilization in this emergent situation.

CASE REPORT

A 60-year-old man with a history of hypertension underwent percutaneous coronary angioplasty followed by stenting of the first diagonal branch (D1) 13 months previously. After experiencing recurrent angina for 3 months, he underwent a repeat coronary angiography evaluation. The cardiac catheterization revealed single-vessel coronary artery disease with a significantly stenotic lesion in the proximal left anterior descending artery (LAD) and the second diagonal branch (D2), and an in-stent restenosis of the D1 (Figure 1). The preoperative transthoracic echocardiogram (TTE) showed a normal ejection fraction without regional wall motion abnormalities.

A median sternotomy was performed, and the left internal thoracic artery (LITA) was dissected and mobilized over its entire length in a skeletonized fashion. The LITA was then wrapped with a papaverine-soaked gauze after topical spray with papaverine. After the pericardium was opened and the relationship between the LAD and the diagonal branches was inspected, 200 U/kg heparin was administered intravenously 3 minutes prior to distal transsection of the LITA, which was a very large conduit with excellent flow characteristics.

Tension on the left pericardial stay sutures and wet gauze pads placed behind the heart facilitated rotation of the entire heart toward the surgeon's side, with the LAD and its diagonal branches becoming near-midline structures.

After confirmation that the length of the LITA was sufficient to reach the LAD, the distal LITA was resected. Then, the resected distal LITA was anastomosed to the proximal in situ LITA in a Y configuration. The distal anastomosis to the coronary artery was done by continuous suturing with single 8-0 polypropylene suture by means of a parachute technique in conjunction with a heart positioner and stabilizer (Starfish and Octopus; Medtronic, Minneapolis, MN, USA). The distal anastomoses were constructed in the following sequence: the proximal in situ LITA to D2 (side-to-side anastomosis), the LAD (end-to-side anastomosis) sequentially, and the resected LITA to D1 (end-to-side anastomosis).

Throughout the procedure, the online-monitored hemodynamics were very stable with acceptable parameter values. After the procedures, graft flow tracing was obtained intraoperatively by means of a transit-time flowmeter (VeriQ system; Medi-Stim, Oslo, Norway) during the hemodynamic stabilization period to demonstrate that the distal anastomoses were patent.

The activated clotting time was kept at >300 seconds, and the heparin effect was fully reversed with protamine at the end of the operation.

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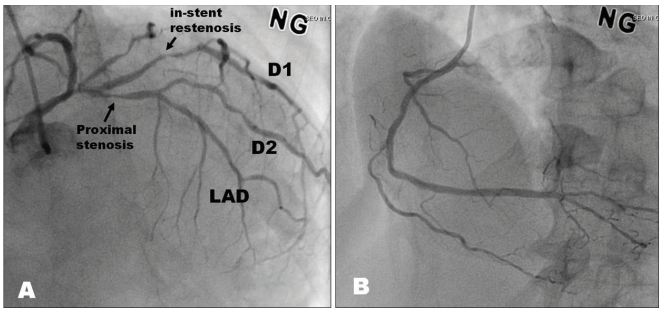

Figure 1. The preoperative coronary angiogram shows stenotic lesions in the proximal left anterior descending artery and an in-stent restenosis of the first diagonal branch. Panel B represents the right coronary artery (RCA) in preoperative angiogram. There was no significant lesions in RCA. D1 indicates first diagonal branch; D2, second diagonal branch; LAD, left anterior descending artery.

The patient was transferred to the intensive care unit (ICU) in stable condition and administered continuous nitroglycerine and diltiazem intravenously. Approximately 5 hours after transfer, the patient developed sudden global ischemia according to electrocardiography, hypotension, and bradycardia, and progressed into sustained cardiac arrest due to ventricular fibrillation. Cardiopulmonary resuscitation, including direct-current electrocardioversion and external cardiac massage, was performed at the bedside in the ICU. Despite resuscitative efforts, hemodynamic and electrical instabilities were sustained. After the intravenous injection of heparin, both the femoral artery and vein were cannulated percutaneously by the Seldinger technique, and PCPS was started with a Capiox emergency bypass system (Capiox EBS®; Terumo Cardiovascular Systems, Tokyo, Japan). An IABP was applied simultaneously. After the initial stabilization with PCPS and IABP, emergent coronary angiography was performed and revealed the patent LITA conduit with severe narrowing of the native coronary arteries (Figure 2). An infusion of high-dose nitroglycerin and sodium nitroprusside into the conduit and native coronary artery relieved the spasm immediately; however, PCPS and IABP supports were maintained because echocardiography evaluation revealed the persistence of left ventricular dysfunction. The patient was administered continuous nitroglycerin and diltiazem intravenously, and nicorandil was added through the nasogastric tube. Follow-up serial TTE evaluations showed progressive recovery of left ventricular function.

After 48 hours, TTE evaluation confirmed significant improvement of left ventricular function with a left ventricular ejection fraction (LVEF) of ≥45% with no segmental wall abnormalities. Successful weaning from PCPS was achieved at this point. PCPS was removed on the second postoperative day, and the IABP was discontinued on postoperative day 3.

The patient was transferred to the general ward on postoperative day 8 and was discharged uneventfully on postoperative day 14 with a normal LVEF.

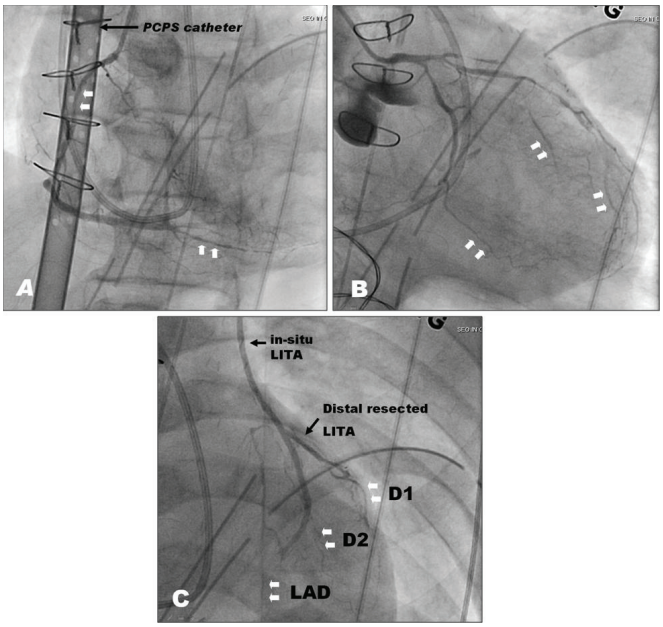

Figure 2. The emergent coronary angiogram reveals diffuse spasm involving the right coronary artery system (A) and the left coronary artery system (B); however, the left internal thoracic artery (LITA) and distal anastomosis sites were intact (C). White arrows indicate spastic coronary arteries. PCPS indicates percutaneous cardiopulmonary support; D1, first diagonal branch; D2, second diagonal branch; LAD, left anterior descending artery.

DISCUSSION

Coronary artery spasm is an unpredictable and serious complication following coronary artery bypass surgery, with an incidence ranging from 0.8% to 1.3% [Buxton 1981; Houppe 1983; Lin 2007]. The incidence and mortality rates may be underestimated, however, because mainly only the surviving cases are reported and because an exact diagnosis can be achieved only with coronary angiography [Zaiac 1990].

The causes of coronary artery spasm after coronary artery bypass grafting have not been fully identified. Several potential factors for postoperative coronary artery spasm have been described, including high endogenous catecholamine levels, manipulation of coronary arteries during dissection, hypothermia, small coronary arteries, withdrawal of intraoperative vasodilators in the early postoperative period, and underlying atherosclerotic disease [Zeff 1982; Lemmer 1988; Paterson 1998; Bittner 2002].

The clinical manifestations following spasm vary, depending on spasm severity or the vessels affected by spasm. In serious cases, coronary artery spasm can proceed to circulatory collapse and death.

Several treatment options for spasm have been reported in case reports. Coronary angiography with infusion of coronary vasodilators (nitroglycerine, verapamil, papaverine, or diltiazem) has been one of the best-established diagnostic and therapeutic tools [Zeff 1982; Lemmer 1988; Paterson 1998; Bittner 2002]. In unstable patients, however, aggressive

cardiopulmonary resuscitation is warranted, and PCPS would be a good tool to resuscitate and stabilize patients initially until an exact diagnosis and prompt treatment with cardiac catheterization.

In our case, the diffuse coronary spasm involved both grafted and nongrafted vessels and led to ventricular fibrillation and cardiac arrest. In this situation, PCPS was a good tool for initial stabilization and as a bridge to early diagnosis and prompt treatment including coronary angiography with infusion of coronary vasodilators. PCPS also contributed to the patient's recovery from left ventricular dysfunction.

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