

Refractory Coronary Artery Spasm after Minimally Invasive Direct Coronary Artery Bypass Grafting

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Postoperative coronary arterial spasm is a rare but potentially fatal complication. A 51-year-old male patient with a history of a reactive ergonovine stress test coronary angiogram developed refractory coronary artery spasm after undergoing minimally invasive direct coronary artery bypass grafting of the left anterior descending coronary artery. The patient was successfully managed with rapid implementation of intra-aortic balloon-pump counter pulsation and extracorporeal membrane oxygenation.

Key words: 1. Coronary artery bypass surgery
2. Assistive devices
3. Vasospasm
4. Myocardial injury
5. Minimal invasive surgery

CASE REPORT

A 51-year-old male patient with a left anterior descending coronary artery (LAD) stent placement 2 years earlier presented with recent chest pains. A follow-up angiogram 1 year after the initial stenting revealed an in-stent restenosis that required stenting. Recurrent chest pains 8 months thereafter were positive for inducible spasm of the left circumflex (LCx) and right coronary arteries (RCA) by ergonovine stress test coronary angiogram leading to a diagnosis compatible with variant angina. In the current admission the coronary angiogram showed a 95% stenotic lesion in the stent comprising a high critical stenosis of the LAD (Fig. 1). As the left ventricle was viable but ischemic without acute myocardial infarction, the patient was scheduled for urgent surgery. The

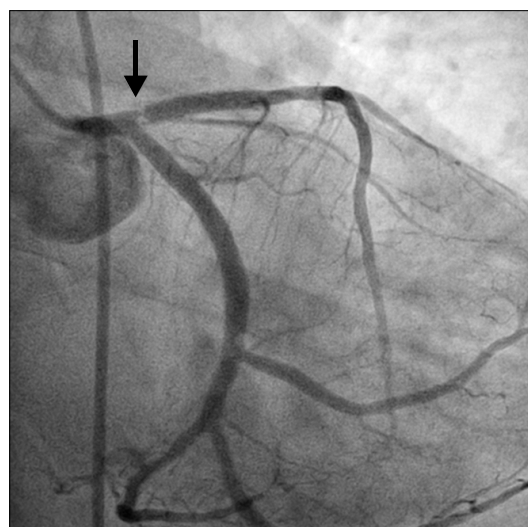


Fig. 1. A preoperative coronary angiography shows critical in-stent restenosis of the proximal left anterior descending artery (arrow).

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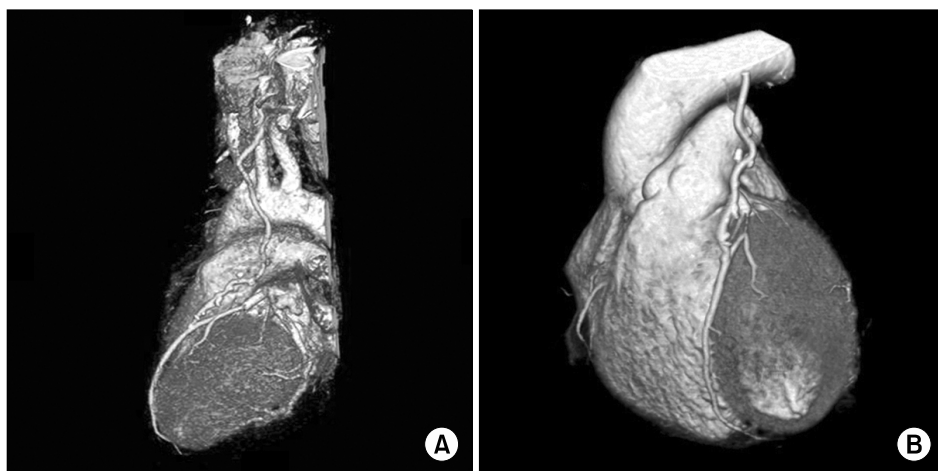


Fig. 3. 64-channel multi-detector computed tomography coronary angiography performed shortly after recovery shows restoration of the left anterior descending artery lumen and excellent patency of the left internal thoracic artery graft (A). Persistent patency and a size increase of the graft is evident 1 year later (B).

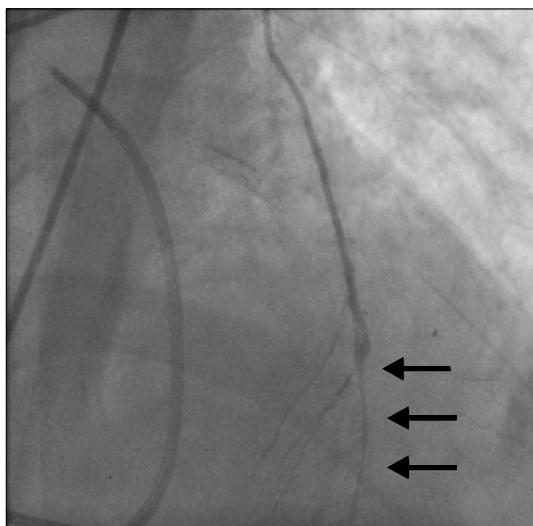


Fig. 2. An immediate postoperative coronary angiography shows severe diffuse spasm of the left native anterior descending artery (arrows) resulting in near total occlusion of the left anterior descending artery lumen.

preoperative echocardiogram at the time revealed an ejection fraction of 57%.

Due to the need to bypass only the LAD, a single left internal thoracic artery (LITA) to LAD bypass was performed by minimally invasive direct coronary artery bypass grafting (MIDCAB). The LITA was harvested under direct vision through a 10-cm left antero-lateral skin incision over the fourth inter-costal space. Given the patient's history of variant angina, on-pump beating heart surgery was performed as a

preemptive measure against the possibility of intraoperative coronary spasm, in which case effective resuscitation would be difficult in the off-pump coronary artery bypass grafting (OPCAB) status. The arterial inflow was established through the left femoral artery and venous drainage through a percutaneous dual stage venous catheter that was passed into the right atrium. The surgery was uneventful and a postoperative transit time flow meter (Medistim BF model; Medistim, Oslo, Norway) showed excellent LITA flow characteristics with a mean flow and pulsatility index of 31 mL/min and 2.2, respectively. However, shortly after transfer to the intensive care unit, the patient developed sudden hypotension and marked precordial ST segment elevation. Immediate intravenous isosorbide dinitrate combined with incremental inotropic support failed to normalize the electrocardiography (EKG) and the hemodynamic condition with worsening of lactic acidosis. Therefore, intra-aortic balloon-pump counter pulsation (IABP) was instituted followed by emergency coronary angiogram. Severe generalized spasm with near luminal obliteration of the native LAD was noted (Fig. 2). However, the integrity of the LITA anastomosis with the LAD was found to be intact. The RCA and LCx arteries were not spastic.

Trans-thoracic 2-dimensional echocardiography at the time revealed an ejection fraction of 30% with LAD territory akinesia, which contrasted with the preoperatively normal left ventricle contractility. Due to the progressing deterioration in patient condition with circulatory collapse, extracorporeal

membrane oxygenation (ECMO) was initiated at 100% support. The cardiac index and cardiac output were maintained at 2.4 L/min/m² and 4,560 mL/min, respectively, until the myocardial function had sufficiently recovered. The CK-MB and Troponin-I levels peaked on postoperative day (POD) 1 at 288 ng/mL and 38 ng/mL indicating perioperative acute myocardial infarction from the culprit lesion. ECMO weaning was possible by POD 4 with near normalization of cardiac enzymes and EKG findings, and improvement in the left ventricle ejection fraction from 30% to 45%. With further improvement, the IABP was removed the following day.

The patient was transferred to the general ward on POD 13. Pre-discharge echocardiography on POD 23 revealed normalized left ventricle function with an ejection fraction of 60% in the absence of discernable wall motion abnormalities. A pre-discharge coronary computed tomography angiogram also showed the LITA graft to be completely patent with resolution of the previous LAD spasm (Fig. 3). The patient was eventually discharged on POD 41 and remained symptom free at 1 year follow-up with a widely patent LITA to LAD bypass graft on computed tomography imaging.

DISCUSSION

Coronary spasm is a well recognized and important cause of postoperative myocardial infarction or stunning. The incidence of medically refractory and angiographically documented coronary artery spasm has been reported to be a rare but potentially fatal complication that may involve either the bypass graft or the native coronary artery [1-4]. It has been implicated in ventricular arrhythmias, cardiac collapse, and other cardiac related deaths [1,4]. Although the exact etiology has not been fully elucidated [3,4], direct coronary artery manipulation, elevated catecholamine levels, hyperventilation, alkalosis, hypothermia, and the release of platelet derived vaso-spastic factors such as thromboxane A₂, autonomic nerve stimulation, and hypomagnesemia have all been cited as possible causes [1,3,4]. Accurate distinction of a spastic from a permanent mechanical cause of coronary occlusion based on symptoms and clinical signs alone may be extremely difficult. Nevertheless, rapid establishment of an accurate diagnosis is key to implementing timely life saving treatment [2,3]. A

strong index of suspicion for coronary spasm based on the patient's prior history of a reactive ergonovine stress test led to a correct diagnosis without unnecessary delays in the present case.

Intravenous or direct coronary ostial infusion of nitrates or calcium channel blockers with intra-aortic balloon-pump counter pulsation in hemodynamically unstable patients is generally accepted as the first line treatment of choice [1-4]. Newer therapeutic agents such as the Rho-kinase inhibitor fasudil [5] or multiple stenting of the spastic coronary artery have also been described as possible treatment options [6]. In the present case, in which all modes of medical treatment were exhausted, intracoronary stent placement would not have been a good option due to the severely stenotic nature of the proximal LAD stent. There was also a high risk of jailing the intact LITA to LAD anastomosis. Therefore, for this patient, IABP and ECMO support with maximal vasodilatory medications was the optimal treatment. To avoid delaying proper treatment of these patients, coronary artery spasm should be suspected in the setting of any new post-operative ischemic complications of unknown cause. Rapid implementation of appropriate treatment for coronary spasm is usually lifesaving and emergency coronary angiogram preceding any decision to undergo re-exploration [2-5] may be critical in preventing potentially fatal delays and unnecessary surgical procedures.

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