

of the heart, a condition called reverse remodeling. Mechanical circulatory support with several devices has been used as a bridge-to-recovery in acute myocarditis for more than a decade [8-10]. Yacoub recently reported the adjunctive effect of ventricular assist devices and the beta2-adrenergic-receptor agonist clenbuterol in patients with chronic, nonischemic dilated cardiomyopathy [11]. Reiss and colleagues [12] from our group also recently reported the successful removal of a Thoratec bi-ventricular assist device (Thoratec Laboratories Corp) in an adolescent with fulminant myocarditis after complete recovery of the left ventricular function. With our weaning protocol, we try to allow enough time for the ventricle to recover under optimized unloading conditions. Based on our clinical experience, this process requires 4 to 6 weeks and is related to each patient's individual condition and capability to exercise.

We believe that this is the first case report in literature of a young teenage boy with cannabis-induced toxic myocarditis treated successfully with LVAD as a bridge-to-recovery. The LVAD was removed successfully 96 days later, after functional recovery of the heart could be demonstrated.

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Intractable LIMA Spasm in the Postoperative Period Treated by Placement of 'Bridging' Stent

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A woman with critical left main stenosis suffered intractable left internal mammary artery spasm post-coronary artery bypass grafting. This was managed with a left main stent and the patient stabilized. A 3-month follow-up angiogram showed a patent graft and a blocked stent.

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Conduit spasm is a recognized complication of coronary artery bypass surgery, particularly effecting arterial conduits. Spasm is multifactorial and there is no consensus on management of severe spasm. We present an illustrative case.

A 64-year-old woman with multiple cardiac risk factors was referred to our unit by the cardiology department after a non-ST segment elevation myocardial infarction. Acute coronary angiogram showed critical left main coronary artery stenosis, mild mid-left anterior descending coronary artery (LAD) disease, severe proximal stenosis of the first marginal and severe ostial right coronary artery (RCA) disease (Fig 1). Due to her critical left main disease, an intra-aortic balloon pump was inserted, and it was arranged to perform urgent coronary bypass surgery. An echocardiogram on the same day showed mild left ventricle (LV) systolic dysfunction and structurally normal cardiac valves.

Emergency myocardial revascularization ($\times 3$) was performed with the left internal mammary artery (LIMA) to the LAD and saphenous vein grafts to targets on the proximal RCA and third obtuse marginal arteries. The LIMA was harvested from the chest wall without incident and was a 2-mm conduit with reasonable free flow. Blood and potassium (4:1 cardioplegia solution) was used, and the LIMA was wrapped in papaverine-soaked gauze after dissection from the chest wall. The LAD was a good target. The patient came off bypass without incident and was transferred to the intensive care unit in stable condition.

However, overnight in the intensive care unit, profound hypotension developed in the patient, which was refractory to high-dose noradrenaline. This was associ-

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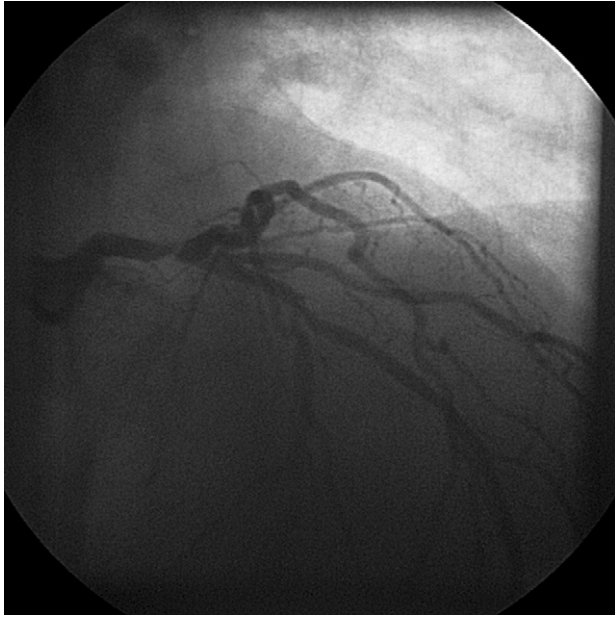


Fig 1. Pre-coronary artery bypass grafting angiogram.



Fig 3. Successful stenting of the left main coronary artery showing flow to the left system.

ated with widespread ST segment depression and T-wave inversion. The troponin peaked at 340 μ g/L.

Emergency coronary angiogram was performed, which showed a completely occluded LIMA graft (Fig 2). This was injected with intra-vessel diltiazem with no effect. The complex proximal left main lesion was then stented as a last resort, and the patient was stabilized (Fig 3). For the next 7 days the patient recovered in the intensive care unit and was discharged.

Follow-up angiogram 3 months later showed an occluded left main stent and a remarkably well patient.

When the LIMA was cannulated it was found to be widely patent and filling the entire left system (Fig 4).

Comment

Spasm of grafted conduit material in the postoperative period is a potentially fatal complication of coronary artery bypass surgery and is usually associated with damage to the vessel at the time of dissection [1, 2]. Other

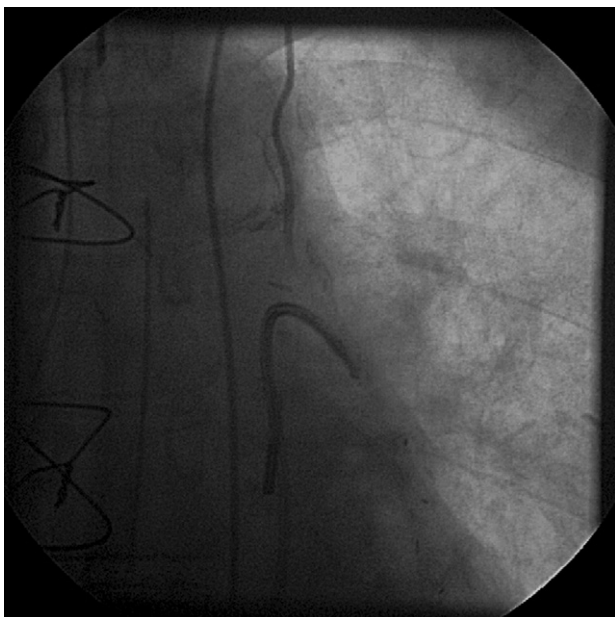


Fig 2. Emergency postoperative angiogram.



Fig 4. Follow-up angiogram with patent left internal mammary artery to the left anterior descending coronary artery.

factors that have been implicated in spasm include stimulation of alpha adrenoreceptors by vasoconstrictor inotropes, alkalosis, and low Pa[SCAP]CO[R]₂, and abrupt cessation of vasodilators, such as calcium channel blockers. Conversely, uses of calcium channel blockers, intravenous nitroglycerine, and local injections of vasodilators (both intraoperatively and by angiographic catheter) have been suggested to reduce the rate and severity of graft spasm and have been used in both a prophylactic and therapeutic setting.

Our search of the literature on this subject revealed several documented cases of treatment of postoperative vasospasm, with injection of intraluminal vasodilators either by angiographic catheterization or by reopening and direct vessel injection [1, 3]. Particularly, nitroglycerine, diltiazem, verapamil, and papaverine have been used. However, only one other case of apparent spontaneous recovery of a vessel in severe spasm was found, and that was in the case of a delayed spasm causing myocardial infarction many years after the original surgery, most likely representing variant angina in a non-native vessel rather than postsurgical spasm [4].

Coronary artery angioplasty and stenting of the graft have been used in the past to treat life-threatening postoperative vasospasm with success [5]. However, it would seem ours represents the only reported case of severe intractable spasm, refractory to intravessel calcium channel blocker, successfully treated with placement of what might be considered a "bridging" stent in a diseased native vessel to allow time for the spasm to reverse on its own accord.

Little is known about the natural history of postoperative coronary vasospasm. Most cases of spasm either go undiagnosed or are successfully curtailed by use of vasodilators, administered systemically or directly through catheterization or at the return to the operating room. Given the rapid reduction of inotropes used after catheterization, and the good recovery made by the patient in this case, we postulate that the LIMA recovered reasonably early during the period between the two angiograms. Certainly, the patient exhibited no symptoms as the left main stent occluded.

This case raises the issue of the extent to which high-dose alpha receptor agonist inotropes contribute to the severity and duration of vasospasm in the postoperative period. It also introduces the novel treatment option of stenting native vessels to manage transient life-threatening conduit spasm, which is not responsive to current conventional techniques.

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Extrinsic Compression of the Left Main Coronary Artery by Atrial Septal Defect

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On rare occasions, extrinsic compression of the coronary artery can cause significant stenosis. We report a 42-year-old woman who was referred to our hospital for surgical repair of atrial septal defect. Cardiac 64-slice multi-detector computed tomography before the operation revealed the extrinsic compression of the proximal left main coronary artery by the marked dilatation of pulmonary trunk. The patient eventually underwent atrial septal defect closure and coronary artery bypass simultaneously. Four months after the operation, multi-detector computed tomographic scan revealed reduction of pulmonary trunk diameter and resolution of left main coronary artery narrowing.

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Coronary artery stenosis is usually caused by atherosclerotic disease. However, on rare occasions, extrinsic compression of the left main coronary artery (LMCA) is caused by pulmonary artery dilatation and the best management is not well defined [1-3]. In this article, we report a 42-year-old patient with atrial septal defect (ASD) who had extrinsic compression of the LMCA by an enlarged pulmonary trunk due to long-standing pulmonary hypertension.

A 42-year-old woman who had been diagnosed with ASD was referred to our hospital for surgical repair. Recently, she had been suffering from progressive dyspnea on exertion, but never experienced chest pain suggestive of typical angina. On physical examination, there were right

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