

The radial artery as a coronary bypass conduit: dealing with hyperreactivity

To the editor: The radial artery (RA) graft is regaining popularity since new data indicates improved graft survival and decreased re-interventions related to the use of arterial conduits. However, radial grafts are known for their high reactivity and propensity to spasm during harvesting and implantation, resulting in early graft failure.¹ Radial artery graft spasm is common during cardiac catheterization.² It is necessary that cardiologists be more familiar with this graft to avoid complications and unnecessary procedures. We report a case of RA spasm during cardiac catheterization, review the literature on RA spasm, and summarize its prevention and treatment.

The patient was a 62-year-old male who underwent coronary artery bypass grafting (CABG) for severe three-vessel coronary artery disease (CAD). He received a left internal mammary artery (LIMA) to the left anterior descending artery (LAD), RA to the first diagonal and a saphenous vein graft (SVG) to the obtuse marginal branch that was skipped to the posterior-lateral branch. A pre-operative myocardial perfusion imaging performed prior to genitourinary surgery demonstrated significant lateral wall ischemia and therefore a diagnostic cardiac catheterization was performed. Angiography demonstrated severe native three-vessel CAD, a patent LIMA, a patent SVG to obtuse marginal branch and occluded jump graft to the pos-

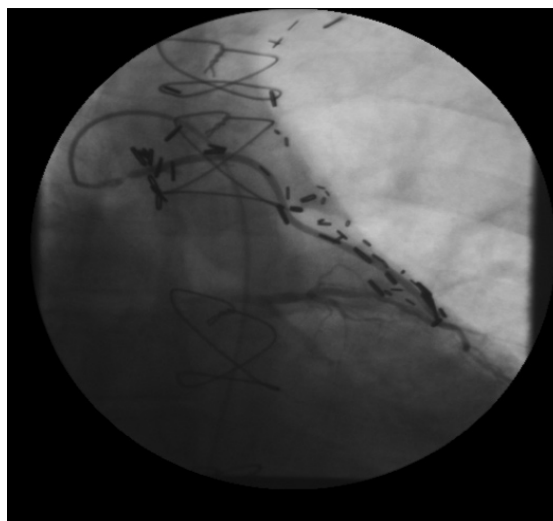


Figure 1. Straight posteroanterior view of the radial artery graft demonstrating the spasm in the proximal end of the graft.

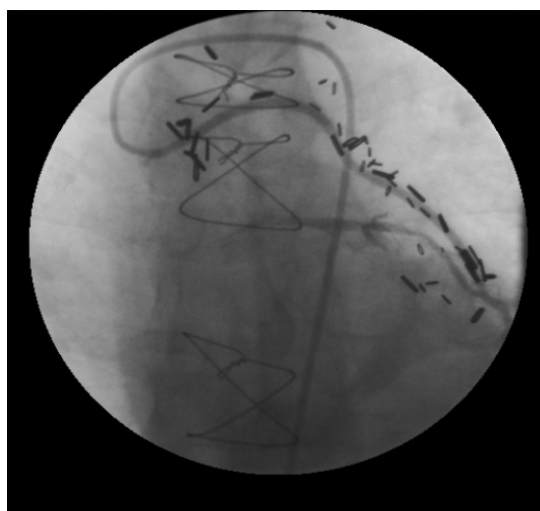


Figure 2. Posteroanterior caudal view showing the partial resolution of the spasm after intracoronary nitroglycerine administration.

terior left ventricular branch. The RA graft demonstrated a severe concentric narrowing proximally (Figure 1). After the diagnostic catheter was exchanged for a guiding catheter and intracoronary nitroglycerine administered, complete resolution of the lesion was observed (Figure 2, 3).

Carpentier first used the radial artery as a coronary graft in 1973¹.

Following the disappointing results from early graft closure related to spasm and late failure from intimal proliferation its use was abandoned. In 1992, widespread use of calcium-channel blockers to prevent spasm resurrected RA graft use,^{1,3} supported by their ability to demonstrate flow-mediated dilatation and resistance to atherosclerosis.⁴ Despite drug therapy, vasospasm still occurs in at least 5% to 10% of radial artery grafts both during and after CABG.⁵

Radial artery hyperreactivity has both a receptor independent and receptor dependent component. In vivo, experiments of radial artery vasoconstriction induced by KCl were resistant to both alpha-adrenergic and endothelin antagonists while abolished by the direct smooth muscle relaxant papaverine. In contrast, alpha-adrenergic and endothelin-mediated spasm was relieved by their respective receptor blockers.^{6,7} Compared to the LIMA, measured strain and tension with vasoconstricting agents KCl, norepinephrine and phenylephrine is higher in radial arteries and pre-treatment with isradipine appears to relieve it to a greater extent, suggesting intrinsic differences in vascular reactivity between LIMA and radial grafts.⁸

In in vivo experiments, norepinephrine-induced constriction was abolished with phenoxybenzamine incubation for 18 hours. However, papaverine pre-treatment inhibition lasted around 8 hours.⁷ Calcium channel blockers exhibit differential responses inhibiting receptor mediated vasoconstriction. Amlodipine and nifedipine significantly ameliorates vasoconstriction compared to diltiazem.⁹

Calcium antagonist and nitroglycerin (NTG) separately abolished up to 80% of vasoconstriction

induced by endothelin-1, while the combination resulted in exaggerated vasodilatation.^{5,10} NTG and nifedipine are more effective than combinations with diltiazem or verapamil.¹¹ These presumably relate to the additional smooth muscle relaxant property with dihydropyridines in addition to receptor inhibition. Studies of long term administration of diltiazem to promote radial artery graft patency have been disappointing.

Large-scale surgical experience in over 6000 patients with radial grafts¹² has demonstrated the safety, low operative mortality (0.9%), sternal infection (1.4%), and myocardial infarction (0.8%) in these patients. Postoperative angiographic RA patency was 90.2% at five years. Internal thoracic artery/radial artery T grafts have been evaluated as sole conduits for myocardial revascularization in two- and three-vessel disease.¹³ Both immediate (99%) and 5-year (90%) survival was improved, as were freedom from postoperative MI (96%), recurrent MI (94%), catheterization (83%) and reintervention (angioplasty or reoperation, 93%).

Long term RA patency appears to be related to target location and proximal target stenosis. In a retrospective evaluation, patency for targets with moderate stenosis (<70%) was worse than that for vessels with critical stenosis (>90%). Also patency for targets in the right coronary was inferior to that of the LAD and the circumflex arteries.¹⁴ Radial artery harvesting and preparation using hydrostatic dilation with 1% papaverine and nicardipine followed by intravenous nicardipine in the peri-operative period¹⁵ demonstrated acute patency of over 95%.

New surgical techniques are being used to reduce early spasm and

graft failure (harmonic scalpel, anti-spasmodic agents). Currently most surgeons prefer T-anastomosis of the radial artery to IMA or vein graft. In addition intra-graft injection of papaverine, nicardipine, no-touch or minimal touch techniques have reduced spasm. Currently adenovirus-mediated gene transfer of eNOS is being evaluated.¹¹ These eNOS transfected segments demonstrate resistance to constriction in-vitro and enhanced responsiveness to nitroglycerine compared to calcium channel antagonists.

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