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Journal

Catheterization and Cardiovascular Interventions, 66(3)

ISSN

1522-1946

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Publication Date

2005-11-01

DOI

10.1002/ccd.20501

Catheterization and Cardiovascular Interventions 66:360–363 (2005)

Coronary Steal Syndrome With Coil Embolization of a Large LIMA Side Branch: Radionuclide Evidence for Reversible Ischemia

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This case presents the controversy over coronary artery steal syndrome following bypass surgery when a large branch of the left internal mammary artery (LIMA) is not ligated. A discussion of previous attempts to understand the physiology of this anatomy is compared with case reports of objective evidence for ischemia that resolves following occlusion of the LIMA side branch. © 2005 Wiley-Liss, Inc.

Key words: left internal mammary artery; coronary bypass surgery; Doppler flow; fractional flow reserve; coil embolization; radionuclide imaging

INTRODUCTION

There is continued controversy whether a large side branch of a left internal mammary artery (LIMA) bypass graft can cause ischemia by "stealing" blood flow from the branch to the left anterior descending coronary artery (LAD) [1]. We address this question by presenting a patient who had objective evidence for ischemia that was resolved following coil embolization of a large LIMA side branch.

CASE REPORT

A 59-year-old woman had three episodes of restenosis over a 1-year period following stent deployment to the mid-LAD. Despite insertion of a drug-eluting stent, restenosis recurred. During the last angioplasty, the guidewire could not cross the stenosis and the patient was sent for single-vessel bypass surgery with a LIMA to the distal LAD. Over the next year, the patient continued to complain of her original symptoms of exertional chest pain and dyspnea. An adenosine Myoview stress test demonstrated mild reversible ischemia in the anteroapical distribution (Fig. 1, left). A repeat angiogram documented the occluded mid-LAD (Fig. 2). The LIMA was patent but there was a large side branch from the main trunk to the chest wall that had not been ligated (Fig. 3). The ejection fraction was 73% with no wall motion abnormalities noted.

A diagnosis of coronary steal syndrome was entertained. The patient was referred to this institution and underwent coil embolization of the LIMA side branch (Fig. 4) using two Cook coils, 0.038″ × 3 mm × 2 cm. After occlusion of the side branch, there was concern whether the proximal LIMA was twisted. A fractional flow reserve (FFR) study was performed and was normal (0.95) in the LIMA. Following the procedure, the patient's symptoms of chest pain and dyspnea resolved. An adenosine Myoview performed 1 month after coil embolization showed resolution of the previous ischemic changes (Fig. 1, right).

DISCUSSION

The LIMA has a large side branch in 10–20% of people [2]. The presence of a large side branch raises concern that blood flow could be diverted away from the anastamosis with the LAD and competitively enter the low-resistance circulation to the chest wall. This argument for a coronary steal phenomena as a cause of post-CABG angina has been raised by many authors.

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Received 23 February 2005; Revision accepted 31 May 2005

DOI 10.1002/ccd.20501
Published online 5 October 2005 in Wiley InterScience (www.interscience.wiley.com).

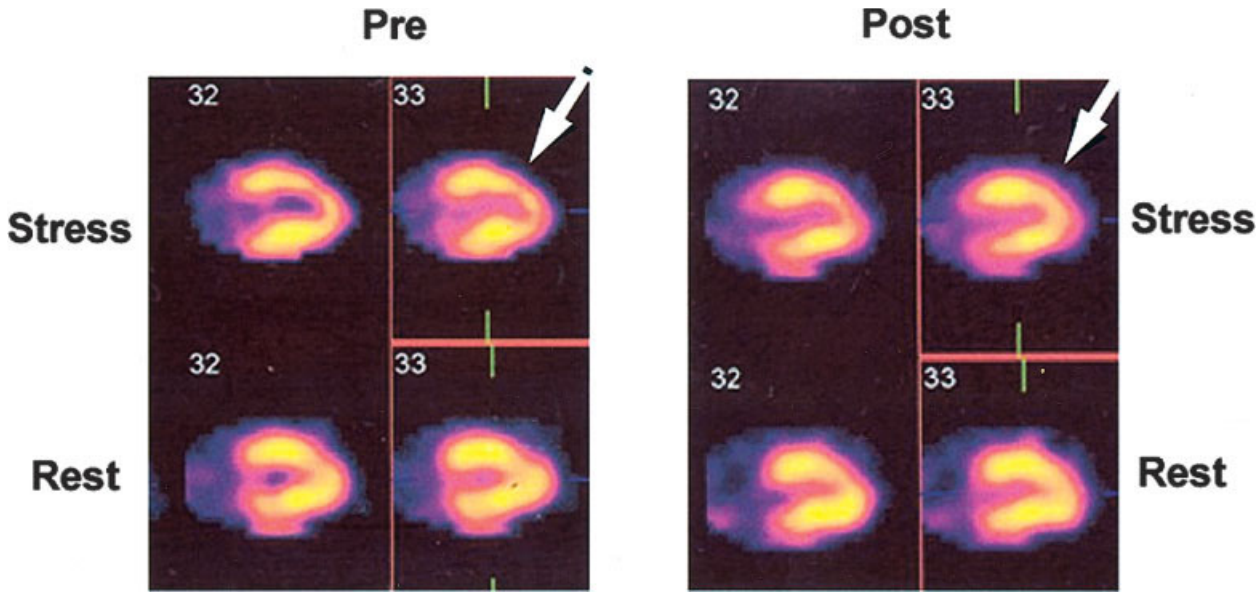

Fig. 1. Left: Radionuclide imaging before LIMA side-branch occlusion. Right: Radionuclide imaging after LIMA side-branch occlusion. [Color figure can be viewed in the online issue, which is available at www.interscience.wiley.com].

Previous studies postulated that a coronary artery steal syndrome is more likely to occur if the side branch is larger than the distal LIMA graft, if there is a stenosis in the distal LIMA anastamosis, or if there is disease progression of the targeted native coronary artery [1–4]. Following open heart surgery, most surgeons ligate the proximal side branches of the LIMA [5], but a recent report in the surgical literature suggests that this might not be necessary [6]. This would be convenient for operators who are performing off-pump procedures, where access to the side branches is limited.

A literature review from 1980 revealed 25 reports involving 43 patients who were identified as having a coronary artery steal syndrome due to a LIMA side branch. These patients underwent side-branch occlusion through various interventions, primarily coil embolization and surgical ligation [1,4]. The clinical outcomes in these reports have been favorable, with 31 of the 43 (72%) patients reported to have clinical improvement following the procedure. Stress tests for ischemia were performed on 15 patients before and after side-branch ligation using different modalities (eight radionuclide imaging, three exercise ECG, and four unspecified). Objective evidence of ischemia prior to the intervention was present in 15 of these cases and 14 had objective resolution of their ischemia following closure of the LIMA side branch [1].

Recently, Kern et al. [1] reported studies in three patients with post-CABG angina and residual LIMA side branches. Doppler velocity measurements were performed at baseline and at peak hyperemia, induced pharmacologically with adenosine and physiologically with exercise. They reported that there was no significant change in LIMA to LAD flow velocity or coronary flow reserve after balloon occlusion of the side branch. This elegant study appears to confirm previous concepts that the LIMA side branch does not divert blood from the LAD since arterial flow to the chest wall through the side branch predominantly occurs in systole and is out of phase with arterial flow of the LIMA to LAD, which is largely dependent on diastolic flow [1,6–10].

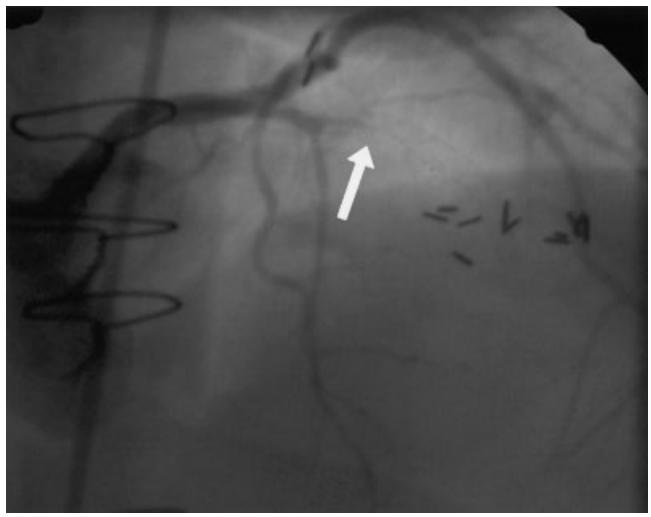

Fig. 2. Angiogram of native LAD occluded in stent after small diagonal branch.

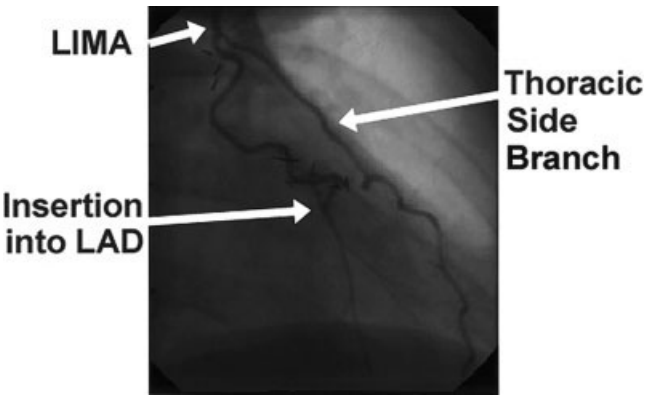

Fig. 3. RAO angiogram of LIMA. Large thoracic side branch with insertion of an equal-sized LIMA into the LAD.

On the other hand, it is hard to ignore the reports of clinical improvement in patients who underwent occlusion of the LIMA side branch. Although the reported clinical improvements could be explained as a placebo effect, it is difficult to discount the objective evidence of reversible ischemia that resolved following LIMA side-branch occlusion in 14 cases.

Kern et al. [1] noted that there were no significant changes in diastolic or systolic flow pattern, or coronary flow reserve in the LIMA after balloon occlusion of a proximal large LIMA side branch. Yet other authors reported significant hemodynamic changes occurring primarily during systole. Luise et al. [6] compared 15 patients with LIMA grafts without side branches to an equal number of patients who had a residual side branch of the LIMA. They reported that the flow pattern in the proximal third of the LIMA was predominantly systolic regardless of the harvesting technique. However, the flow pattern in the distal third of the LIMA showed significant differences between the two groups. The diastolic-to-systolic peak velocity ratio (DSPVR) was higher in the group of patients with the LIMA side branches ligated compared to the group with patent LIMA side branches (1.7 ± 0.1 vs. 0.97 ± 0.3: $P < 0.0005$). There was no significant change in coronary flow reserve following induced hyperemia in this study, although the dose of adenosine (12–14 μg) may not have been sufficient to mimic physiological hyperemia.

Furthermore, Abhyankar et al. [7] reported a case where Doppler flow in the LIMA demonstrated systolic retrograde blood flow that resolved after balloon occlusion of the LIMA side branch. This systolic reversal is unusual unless steal is present [11]. It also suggests that systolic flow to the myocardium may be important.

By analogy, this is similar to the question whether a myocardial bridge can cause ischemia when the coronary artery is compressed by the myocardium during systole [12]. Ge et al. [12] studied intracoronary ultrasound imaging and Doppler flow patterns in 61 patients with angiographic evidence for myocardial bridging. They reported that systolic compression of the bridge segments is associated with a decrease or

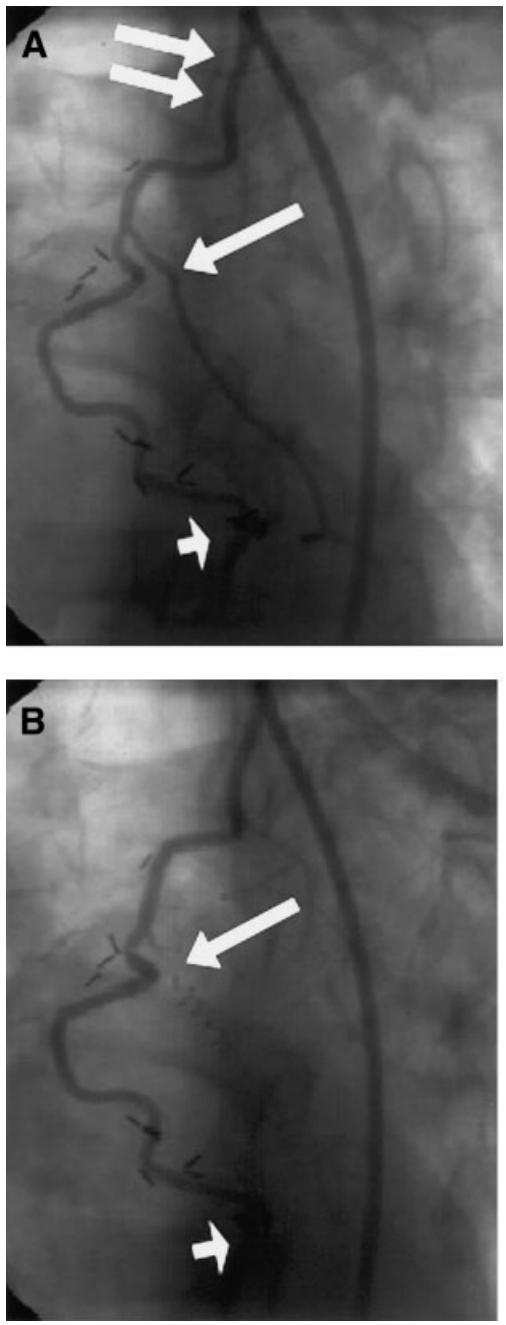

Fig. 4. A: LAO angiogram shows a large LIMA side branch prior to treatment. Double arrow denotes proximal LIMA; long arrow, thoracic side branch; short arrow, LIMA insertion to LAD. B: Repeat angiogram after LIMA side-branch coil embolization. Long arrow denotes thoracic side branch with coils; short arrow, LIMA insertion to LAD.

retrograde systolic flow in the segment proximal to the bridging. This implies that altered systolic flow may be a component of ischemia. Some of the differences in previous reports could be explained by variability in the anatomy, where only the largest side branches may cause enough diversion of systolic blood to produce ischemia.

We believe that Doppler velocity measurements are insufficient to resolve this question. These catheters do not measure blood flow and they do not indicate whether the amount of blood perfusion to a myocardial bed is adequate. Although Kern et al. [1] disparage radionuclide imaging in their article, there is no consensus that Doppler flow is a more accurate gold standard in diagnosing ischemia. It can be argued that SPECT or PET imaging is a more objective measure of perfusion deficits, especially if they resolve following occlusion of the side branch.

This clinical condition is too rare to lend itself to a randomized controlled trial. However, this case report may serve to stimulate the controversy that this clinical entity may be a valid cause of chest pain following coronary artery bypass surgery. We believe that it is still reasonable to occlude the LIMA side branch if objective evidence for ischemia is documented. A registry of these case reports could act as a data bank that might help identify criteria when it is appropriate to perform ligation or coil embolization of the LIMA side branch.

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