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Coronary Steal Due to Left Internal Mammary Artery Side Branch – A Dilemma

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Coronary Steal Due to Left Internal Mammary Artery Side Branch – A Dilemma

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INTRODUCTION
Internal Mammary Artery (IMA) is the best graft for Coronary Artery Bypass Graft surgery (CABG) in terms of long-term survival rates and graft patency rates when compared to other grafts. In traditional CABG, IMA is harvested from 1st inter-costal space and is skeletonized by ligating all the side branches, which is usually 10 cm long. In 1995, Benneti introduced Minimally Invasive Coronary Artery Bypass Graft surgery (MIDCAB) for various benefits. In this technique Left IMA (LIMA) is harvested from 5th inter-costal space and long enough to reach Left Anterior Descending Artery (LAD) around 4-6 cm. The later technique is used mostly for single vessel bypass and off-pump CABG. It is possible that with increased use of this technique the unligated side branches of IMA could lead to coronary steal phenomenon giving post-CABG angina for which no other explanation is available.

Coronary steal syndrome secondary to subclavian stenosis proximal to LIMA bifurcation is well described. A possibility of coronary steal phenomenon secondary to proximal large side branches of LIMA have been reported in literature. These reports have proven the existence of steal phenomenon due to large secondary branches of LIMA on the basis of subjective evidence of angina free periods and documentation of occluded side branches. In 2005, Abdo ET. al., demonstrated radio nucleoside evidence of reversible ischemia and improvement after embolic occlusion of side branches, while in 2004 Guzon et al., demonstrated no significant difference in coronary artery blood flow during rest or adenosine dilation after occlusion of side branches. Hence, the topic of coronary steal phenomenon due to side branches of IMA is inconclusive and review of existing literature is essential.

CASE PRESENTATION
A 44-year-old African-American male who had a single vessel coronary artery bypass surgery LIMA-LAD after being found to have in-stent thrombosis in the proximal LAD that was placed about a month ago. The patient had a history of recurrent exertional angina and poor exercise tolerance despite maximal medical therapy over the preceding 12 months. As a part of work up, a nuclear stress test was performed which showed inducible ischemia in the anterior wall distribution. Cardiac catheterization showed a high-grade lesion in the distal part of the LIMA, for which he received a drug-eluting stent. He also had a patent unclipped LIMA side branch going to the chest wall which was seen on cardiac cath.

As the patient continued to have angina symptoms even after distal LIMA stenting, a persistent intercostal side branch was thought to be the cause of coronary steal. Cardiac catheterization was performed. After standard preparation femoral artery access was gained and 5 Fr internal mammary catheter was used to engage the LIMA graft. Visualization of the graft revealed patent stent, TIMI-3 flow with persistent unclipped side branch. Then a PT2 intracoronary guide wire was passed through LIMA and distally in big unclipped side branch of LIMA. A micro-catheter was then passed distally to the guide wire near the chest wall and two 4 x 20 mm tornado coil by Cook were used and successful coil-embolization was performed without any flow. The patient at one month follow-up visit reported reduced angina and increased functional capacity.
DISCUSSION

LIMA to LAD conduits are used most frequently and known to cause myocardial ischemia owing to incomplete revascularization, graft failure, stenosis at anastomotic site, progression of disease distal to anastomosis, subclavian artery stenosis, congenital anomalies of the brachiocephalic vessels, and coronary steal via a large unligated intercostal, pericardial, or bronchial side branches. Incidence of anomalous or large IMA side branches has been reported to be 9-25% in general population. Although only a few of them develop myocardial ischemia secondary to side branch steal phenomenon. Many case reports have been published showing LIMA side branches causing steal phenomenon with improved subjective and objective outcomes after side branch occlusion. In many of these cases, objective evidences were limited to improvement in ejection fraction on echocardiogram or change in ischemia shown on stress test.

It is well established in at least two case reports that progressive native vessel disease in LAD and anastomotic site stenosis can cause IMA run-off if side branches are present and significant improvement in IMA diameter after correcting the stenosis at anastomotic site. In 2005, Raval et al., demonstrated improved coronary flow reserve (CFR) from 2 to 3.3 and improved ejection fraction from 36% to 70% after occlusion of the side branch by coil embolization. There exists some evidence that supports the theory of coronary steal due to side branches of LIMA, but substantial objective evidence of improvement in ischemia is lacking. In 2004, Guzone et al., demonstrated in his case series with traditional bypass surgery LIMA CFR and average peak velocity (APV) did not change significantly on occlusion of side branches during rest or adenosine induced hyperemia. In 1997, Abhayankar et al., showed minimal increase in APV from 5.6 to 5.8 m/s without any change in LAD diameter on temporary occlusion of side branch of LIMA with balloon.

Finally, permanent occlusion was not performed and patient reported subjective improvement. There are two approaches for bypass surgery, a traditional CAB and MIDCAB. In later the side branches of LIMA are not ligated. It has been postulated that this could give rise to possible steal phenomenon. Physiologically LAD flow is diastolic and flow in LIMA its side branches is systolic. After the bypass, this physiology is slightly altered however still the flow in LAD remains diastolic and diastolic- systolic peak velocity ratio (DSPVR) is measured to assess optimal coronary flow. In 1997, Luise et.al., evaluated the impact of this MIDCAB technique versus the traditional CABG.

Two groups with 15 patients each with different approach of LIMA to LAD conduit were selected with similar non-atherosclerotic profile of the conduit after a post-operative period of approximately 79 days. Flow velocity and LAD diameter were measured at rest and with adenosine injection. The DSPVR is considered normal when it is around 2. In traditional CABG group DSPVR in distal third of the conduit was 1.72+0.1 and that in MIDCAB group was 0.97+0.3 with a p value of <0.0005, although the peak velocity at rest and after adenosine injection in both the segments (proximal and distal part of the conduits) in both groups had no significant difference. This difference of reduced diastolic flow in the MIDCAB group at an average of 80 days could further change with increasing post operative period; however no studies have been done to evaluate that hypothesis. In our case and in many other cases like Raval et.al., the traditional CABG was performed but had unusually large side branches which could have affected the DSPVR giving significant angina and improvement following the occlusion.

Persistent angina in patients with patent LIMA-LAD and native LAD post graft DSPVR, APV at rest and hyperemia and change in LAD diameter with side branch occlusion should be evaluated to determine potential ischemic territories before performing permanent occlusion of the side branches. Meanwhile local methods of CABG, availability of functional studies to evaluate grafts and knowing the existence of either anomalous LIMA side branch or unligated LIMA side branch can further individualize treatment plan. More studies comparing the two techniques and flow dynamics in the conduits after longer post operative period is necessary to further decipher the controversial side branch steal phenomenon.
References:


