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Case Report

Rare case of proximal coronary plaque regression after distal arterial grafting

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ABSTRACT

The metabolically active endothelium of arterial grafts is reported to slow the atherosclerotic process in the distal coronary territories to which they are grafted with improved patency. The literature on arterial grafting causing proximal plaque regression is scant. We report here a case of proximal left anterior descending artery (LAD) plaque regression following distal arterial grafting with a left internal mammary arterial (LIMA) graft to LAD. This rarely documented regression resulted in "stringing" of the arterial graft, while there was a significant progression of atherosclerotic disease in the right coronary with patent venous graft and de novo lesion in circumflex territory necessitating percutaneous coronary intervention, in spite of aggressive medical therapy. The dichotomous progression of disease in two out of three coronary arterial systems and regression of the proximal plaque in LAD grafted with LIMA 12 years ago suggests the protective effect of arterial grafting in reversing the atherosclerotic process.

1. Introduction

Coronary plaque regression is facilitated by aggressive reductions in LDL and increases in HDL levels which form the therapeutic goals of statin therapy.[1] Coronary artery bypass grafting (CABG) utilizing arterial grafts has been documented to retard the atherosclerotic disease progression in the distal coronary territories. The release of vasoactive molecules from the endothelium of the arterial grafts has been proposed as the probable mechanism behind distal plaque regression.[2] However, the regression of proximal coronary plaques with distal arterial grafting has been reported only once.[3] Here, we report a case of regression of proximal left anterior descending artery (LAD) plaque 12 years after CABG with a pedicled, in situ, left internal mammary artery (LIMA) graft.

2. Case report

A 67-year-old hypertensive, dyslipidemic male presented with recent onset recurrent Canadian Cardiovascular Society (CCS) class II angina on exertion (AOE) for past 2 months. He had previously undergone off pump coronary artery grafting (OPCAB) for critical double vessel disease (diagnosed following evaluation for AOE-CCS class II) 12 years ago. The preoperative exercise testing revealed significant ST segment depression with delayed recovery in the anterior, septal, and inferior leads

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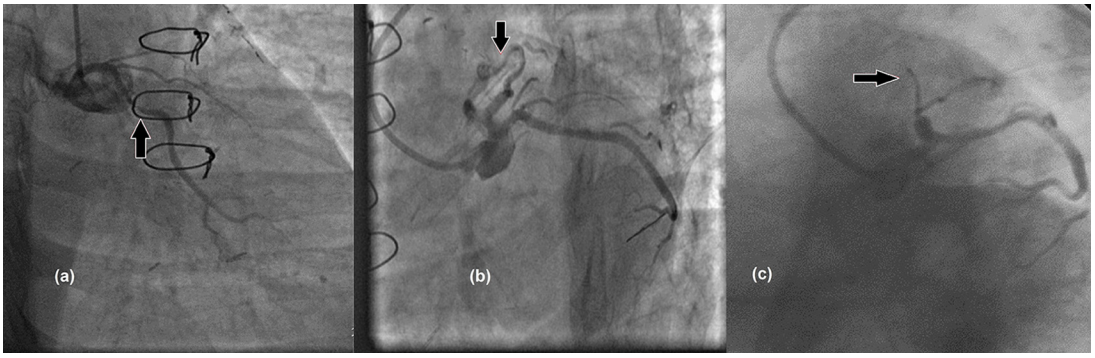

Fig. 1 – (a) De novo lesion in LCx (arrow). (b) Successful PCI to LCx lesion-Flow in LAD (arrow) to be noted. (c) Patent LCx prior to OPCAB with flow limiting lesion in LAD (arrow) 12 years back.

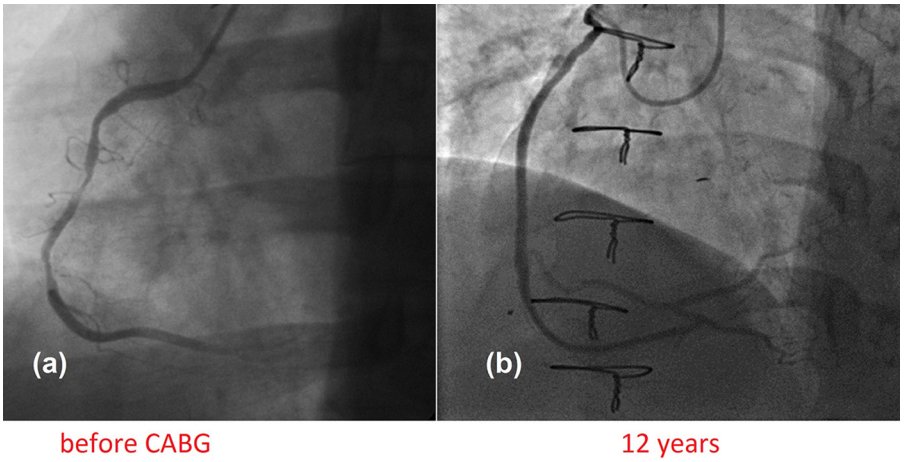

Fig. 2 – (a) RCA disease prior to OPCAB. (b) Patent RSVG graft with progression to CTO of RCA lesions.

and he subsequently underwent coronary angiogram (CAG), which revealed critical stenosis involving proximal LAD (80%) and 70% and 80% tandem stenosis of the proximal and mid right coronary artery (RCA), respectively. He underwent OPCAB with a pedicled LIMA to LAD graft and a reversed saphenous vein graft (RSVG) to posterior descending artery (PDA). He had no other comorbidities or risk factors for coronary artery disease (CAD) and was compliant with an optimal drug therapy, which consisted of aspirin, statins (10 mg of Simvastatin initially after surgery which was subsequently increased to 20 mg for the last 8 years and to 40 mg for the last 2 months), ACE inhibitors, and beta-blockers during the follow-up period. He was compliant to the CAD medical therapy and adhered to a moderate intensity physical activity regimen, which involved 30–40 min of walk for 6 days a week. He remained in CCS class I for the past 12 years with recent onset of recurrent class II effort angina. He had preserved LV function (no regional wall motion abnormalities, ejection fraction of 60% with grade I diastolic dysfunction) and was stage II treadmill test (TMT) positive. CAG done revealed de novo culprit lesion in proximal circumflex territory (LCx) which was treated by percutaneous coronary intervention (PCI) with drug eluting stent (DES), with resolution of symptoms (Fig. 1(a)–(c)). RSVG to PDA was patent with progression of the lesions in RCA to chronic total occlusion (CTO) (Fig. 2(a) and (b)). LIMA angiography revealed stringing of the arterial graft (Fig. 3). Interestingly, a regression of the original lesion in the proximal LAD was noted with re-establishment of an unobstructed

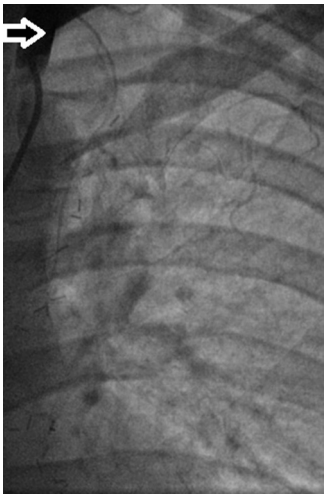

Fig. 3 – Stringed LIMA (arrow).

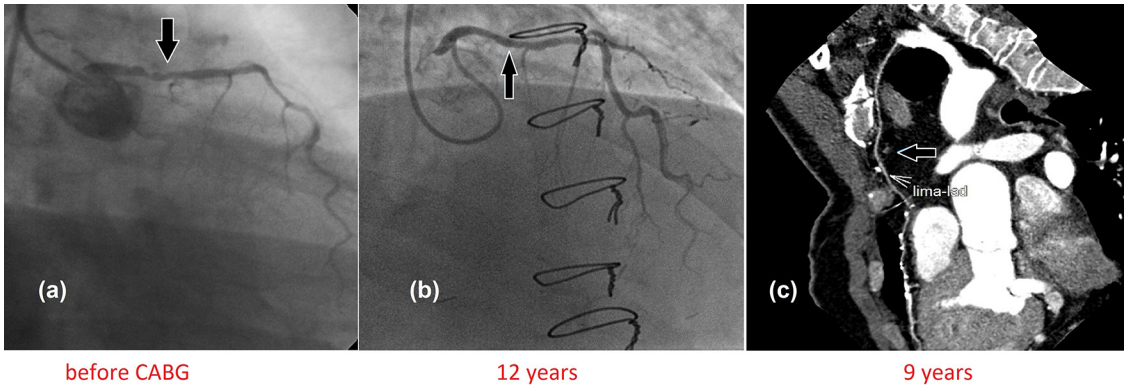

Fig. 4 – (a) Critically diseased proximal LAD prior to OPCAB (arrow). (b) Plaque regression in LAD (arrow) with increased flow in the septal arteries. (c) CTCA demonstrating free flowing LIMA graft with no stringing at 9 years follow-up (arrow).

antegrade flow (TIMI 3) in the artery which explained the competitive flow related stringing of LIMA graft (Fig. 4(a) and (b)). This observation was further corroborated by a computed tomography coronary angiography (CTCA) performed 9 years after the surgery prior to an inguinal hernia repair demonstrating a patent and free flowing LIMA-LAD graft (Fig. 4(c)). At the time of reporting, the patient remains asymptomatic and is free from inducible ischemia as evidenced by his recent TMT. The rare finding of proximal plaque regression following arterial grafting makes this case unique and worth reporting as it reinforces the advantages of arterial grafting.

3. Discussion

Coronary atherosclerosis is a dynamic pathology, which usually follows a progressive course. Plaque progression is a multicentric process affecting different arterial beds simultaneously.[4] This process is retarded by the use of aggressive lipid lowering strategies. The favorable, statin-induced alteration in the lipid profile retard and at times, regress the coronary plaques as concluded by recent studies like SATURN.[1] The culprit lesion in LCx explained the recurrence of his symptoms after 12 years, which was treated effectively by PCI-DES. In this particular case, although the patient had been adhering to healthy life style modifications and an aggressive medical regimen which included high dose statin therapy along with hypertension control and regular physical activity, the disease progression in the LCx and RCA coronary beds could not be modified, as evidenced from the de novo lesion and CTO respectively in these territories.

''String Phenomenon'' or ''disuse atrophy'' of LIMA was first described by Barner as early as in 1974 and was proposed to be due to the competitive flow in the native coronary arteries, to which it is grafted.[3,5] The major causes implicated are flow competition secondary to the grafting of LIMA onto less than critically occluded coronary territories, harvest injuries, spasm, and steal phenomenon resulting from a large undivided proximal branch.[6] The classical ''distal stringing'' seen in this case, could be attributed to the proximal plaque regression and the consequent flow competition.

Arterial grafts, especially LIMA and radial arteries are known to retard the disease progression in the distal coronary territories as evidenced from the MASS II trial and other studies.[2,7] The metabolically active endothelium of arterial grafts produce nitric oxide, vascular endothelial growth factor (VEGF) molecules, and endothelial progenitor cells which may protect the coronary bed to which they are grafted from atherosclerosis in addition to ensuring their own patency.[8] CABG performed on a critically occluded coronary system results in both antegrade and retrograde flow across the vessel lumen. This potentially could subject the proximal plaque to the beneficial effects of arterial grafting and subsequently effect plaque regression. The progression of the disease in other coronary beds (LCx and RCA), devoid of the protective effects afforded by an arterial graft probably, resulted in critical lesions in these territories in spite of adherence to an optimal and aggressive medical therapy.

The critical nature of the lesion (Fig. 4(a)) was evident on the CAG at the time of index surgery, in the setting of class II AOE and the corresponding stress induced ischemia. Fractional flow reserve (FFR) was relatively a newer, less popular, and expensive technique lacking well-defined recommendations for its use 12 years ago. The adequacy of OPCAB, optimal postoperative medical therapy and the long term patency of the grafts (LIMA and RSVG) are supported by the nonrecurrence of angina after OPCAB and the symptom free follow-up for 12 years. The CTCA done 3 years ago showing a free flowing LIMA graft without any evidence of stringing validates the decision to graft LAD in retrospect.

4. Conclusions

Proximal plaque regression after, distal arterial grafting thus, is a rare but a plausible entity, which should be carefully investigated, in a larger cohort of patients. The clinical data presented here favor multiple arterial grafting strategy in critically stenosed vessels for its potential to modify the coronary atherosclerotic process.

Conflicts of interest

The authors have none to declare.

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