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CASE REPORT

Obscure Rare Cause of Recurrent Angina in Post CABG Patient: Coronary-Subclavian Steal Syndrome

I. S. Monga[1,*] and M. K. Yadav[2]
[1]*Senior Advisor (Medicine & Cardiology), Interventional Cardiologist Command Hospital (Eastern Command), Kolkata, India*
[2]*Resident (Medicine), Command Hospital (Western Command), Panchkula, India*

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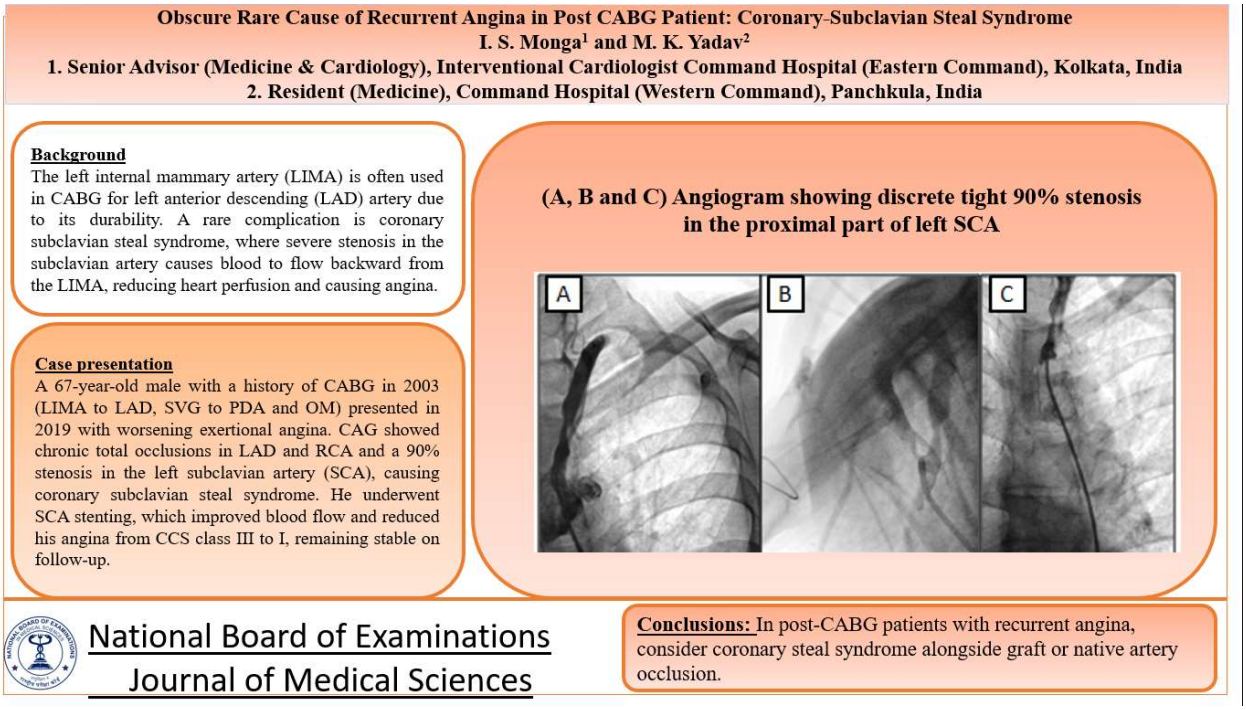
Abstract
Coronary subclavian steal syndrome (CSSS) is a rare and unrecognized complication of coronary bypass grafting surgery when a left internal mammary artery (LIMA) graft is utilized for left anterior descending (LAD) artery revascularization without knowing left subclavian artery status. We describe a case of a 67-year-old male, with a known history of triple vessel coronary artery disease (CAD) managed with CABGx3 in March 2003. The patient presented after 16 years with complaints of recurrent angina for the past 15 years which aggravates on exertion, especially of left hand. Recurrent hospitalizations and repeated Coronary angiograms (CAG) as well as CT Coronary angiography failed to diagnose the cause of angina. Subclavian angiography and subsequent methodical clinical examination during the present admission revealed left subclavian origin stenosis which was successfully revascularized by deployment of a stent resulting in convincing relief in the patient's angina.

Keywords: Coronary subclavian steal syndrome, left internal mammary artery, coronary artery bypass graft surgery, coronary artery disease

*Corresponding Author: I. S. Monga
Email: docinder_86123@rediffmail.com

Graphical Abstract



Introduction

The left internal mammary artery (LIMA) is frequently used as a graft for left anterior descending (LAD) artery revascularization during coronary artery bypass graft surgery (CABG) because of long-term graft survival and superior patency rates [1]. Rare complications of CABG surgery with LIMA as the graft is coronary subclavian steal syndrome. It is characterized by severe stenosis of the proximal subclavian artery (SCA) which results in decreased myocardial perfusion due to retrograde blood flow from LIMA to the proximal subclavian artery for maintaining perfusion of upper extremity. This 'stealing' of blood by retrograde flow to subclavian artery through LIMA induces ischemia and results in angina due to compromised myocardial perfusion.

Case Presentation

We describe a case of a 67-year-old male, reformed smoker with a history of hypertension and triple vessel coronary artery disease (CAD) with a history of CABGx3 in March 2003 (LIMA to LAD and SVG to PDA and OM) who presented to cardiology OPD at this hospital on 01 Oct 2019 with moderate intensity left sided recurrent anginal chest pain since 2004 which aggravates on exertion, especially with left hand. For the initial year after CABG patient was right when he developed angina on exertion which was managed conservatively with anti-anginal medications with some symptomatic relief. However, he continued to be symptomatic with gradually progressive exertional angina and presented to the cardiac center in June 2011. His check coronary angiography (CAG) revealed complete total occlusion (CTO) of mid-LAD, proximal right

coronary artery (RCA), and patent LIMA and reversed saphenous vein graft (rSVG) to PDA and OM. His 2D Echocardiography revealed a left ventricular ejection fraction of 45%. So he was continued on antianginal drug therapy only. He was again admitted in Jan 2017 with similar complaints of worsening exertional angina and a check CAG (04 Jan 2017) again revealed CTO of LAD and RCA, 50-60% lesion in proximal left circumflex artery (LCX), 50% lesion in the proximal ramus, ostial LMCA plaque with patent LIMA to LAD and blocked rSVG to PDA and OM from ostium. The patient was therefore sent for CT Coronary angio before deciding on any further invasive treatment. Coronary CT angiography showed patent LIMA to LAD graft with normal caliber and adequate distal opacification and non-opacification of rSVG graft to PDA. Thereafter he was sent for stress myocardial perfusion imaging (MPI) with ^{99m}Tc MIBI (Feb 2017) showing fixed perfusion defect in the apex, adjoining septal and inferior wall, and left ventricular dysfunction which was aggravated on stress. He was still kept on maximal medical management siting fixed perfusion defect in RCA and OM territory. However, he continued to have progressive symptoms despite maximal medical therapy and reported to cardio OPD on 01 Oct 2019. This time we admitted him for check CAG given severe anginal symptoms despite maximal medical therapy.

Routine hematological and biochemical investigation revealed elevated serum urea (69 mg/dL), and creatine (1.96 mg/dL). Total cholesterol- 255 mg/dL (LDL 172 and triglyceride- 210 mg/dL), CKMB- 28 IU/L and LDH- 513 U/L. Urine routine and microscopic tests were within normal range. His USG KUB revealed a small-sized left kidney with increased cortical echogenicity with loss of cortico-medullary differentiation. Electrocardiogram (ECG) revealed sinus bradycardia, QS in V_1-V_4, T wave inversion in I, aVL, and V_2-V_6(no new ST or T wave changes compared to previous ECG). 2- D echocardiography revealed left ventricular ejection fraction (LVEF) of 45% with anterior wall hypokinesia, concentric left ventricular hypertrophy (LVH) and mild mitral regurgitation.

CAG was done after contrast-induced nephrotoxicity (CIN) prophylaxis on 03 Oct 2019 which revealed CTO of mid-LAD and distal-RCA, 50% plaque in proximal ramus and ostial LMCA (Figure 1A), patent LIMA to LAD (Figure 1B) and complete ostial occlusion of rSVG to PDA and OM (absolutely no change in status since CAG done in Jan 2017). However, after profiling of LIMA, as the diagnostic JR catheter was being pulled back into the aorta, a highly significant pull-back gradient ≥ 90 mm Hg was noted at the origin of the left SCA. Further profiling of the left SCA with the same catheter at the ostia of Lt SCA revealed discrete tight 90% stenosis in the proximal part of the left SCA (Figure 2). Therefore, this patient was now found to be suffering from focal stenosis at the first part of Lt SCA as the cause of his recurrent angina and was planned for peripheral angioplasty of Lt SCA at a subsequent date. Detailed physical examination was carried out which was probably missed during all previous OPD or IPD visits. Physical examination revealed a feeble left radial and brachial pulse. An inter-arm blood pressure difference of 95 mmHg

systolic (187/85 mmHg in the right arm and 92/53 mmHg in the left arm) was present. However, no supraclavicular bruit could be heard. On direct questioning, the patient also gave a history of an increase in angina pain while using the left upper limb.

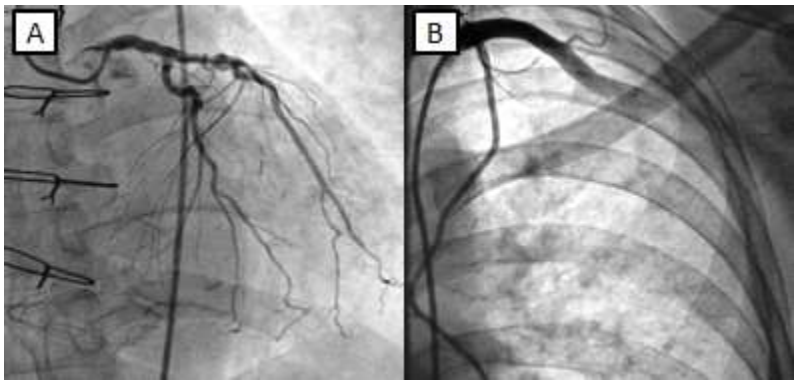


Figure 1. Coronary angiogram showing CTO of mid-LAD and distal-RCA filling retrogradely, 50% plaque in proximal ramus and ostial LMCA (A), patent LIMA to LAD (B)

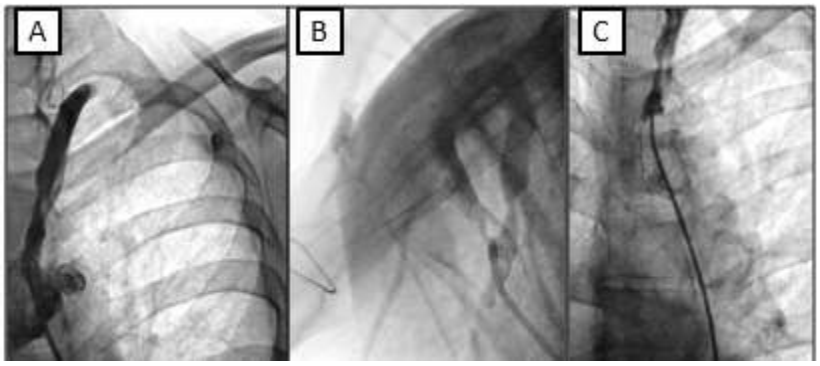


Figure 2. (A, B and C) Angiogram showing discrete tight 90% stenosis in the proximal part of left SCA

Thus, our patient was diagnosed with coronary subclavian steal syndrome as a rare and obscure but treatable cause of recurrent angina among patients who had undergone CABG in the past. He was therefore managed by left subclavian artery stenting on 10 Oct 2019 at our hospital with an 8x37 mm scuba BMS stent (Figure 3). Post procedure good flow was achieved across the stent and through LIMA graft. The patient also showed symptomatic improvement from CCS class III to CCS class I post-procedure. He was on regular follow-ups at 1, 3, 6, and 11 months and continues to be in CCS class 1 despite reducing his anti-anginal therapy.

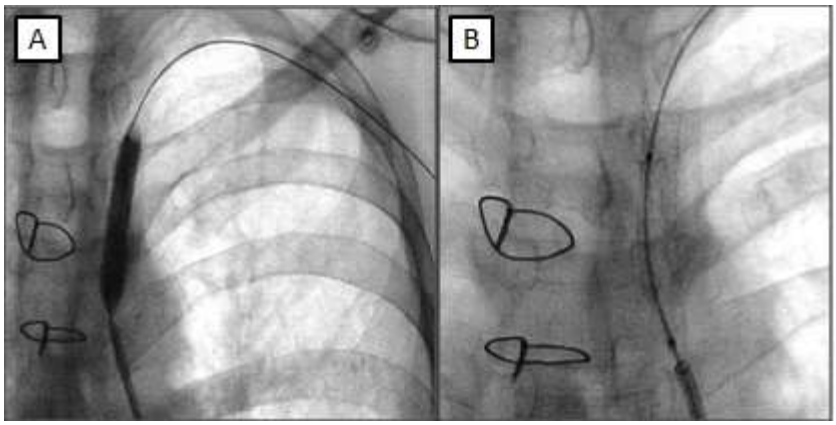


Figure 3. (A and B) Left subclavian artery stenting with 8x37 mm scuba BMS stent

Discussion

Coronary subclavian steal syndrome (CSSS) is caused by atherosclerotic stenosis of the proximal part of the subclavian artery before the LIMA graft. Reversal of blood flow from LIMA to left SCA results in myocardial ischemia in post-CABG patients [2]. Atherosclerosis of native coronary vessels and disease in the graft vessels are the main causes of angina in post-CABG patients. Compromised myocardial perfusion due to CSSS should be kept in differentials for angina in the post-CABG patient [3]. Harjola and Valle first described coronary subclavian steal syndrome in 1974 [4].

Although CSSS commonly presents as recurrent episodes of angina after upper limb stress it may be asymptomatic occasionally and can manifest with a myocardial infraction, heart failure, silent ischemia, vertebrobasilar insufficiency symptoms such as dizziness, ataxia, syncope, blurring vision, ataxia, numbness, drop attacks and upper extremity claudication. Individuals have been reported to present with symptoms between 2-31 years following intervention and presentation within a year following CABG is suggestive of the missed stenotic lesion during initial surgery [5]. Various causes of CSSS include ipsilateral subclavian artery stenosis, Takayasu arteritis, radiation arteritis, and hemodialysis AV fistula [6,7]. The various risk factors for CSSS include advanced age, smoking, hypertension, diabetes mellitus, and non-subclavian vascular calcification [8,9]. CSSS should be suspected in an individual with a history of peripheral vascular disease associated with the presence of more than 20 mmHg inter-arm pressure difference [10]. Noninvasive imaging modalities for CSSS include duplex ultrasonography of supra-aortic vessels, computed tomography and MRI. Proximal aortic arch arteriography is gold standard for the definitive diagnosis of CSSS which shows the presence of flow reversal, including complete retrograde flow in the LIMA.

CSSS can be avoided by using vein conduits and radial artery conduits for CABG. Surgical and radiological-guided endovascular procedures are available methods for the management of CSSS. Preoperatively detected SCA stenosis can be combined with direct subclavian artery bypass surgery. Post-operatively diagnosed

SCA stenosis should be managed with percutaneous angioplasty and stenting like in our case. The radiological endovascular revascularization approach with percutaneous transluminal angioplasty (PTA) and peripheral stenting is the first line of treatment for SCA stenosis [11,12]. These radiological procedures do not require general anesthesia, are minimally invasive, have a shorter hospital stay, and decrease morbidity and mortality [11,13] when compared to surgical bypass techniques. Recurrent stenosis rate is low with stenting as compared to angioplasty [13,14]. In patients with complete occlusion of SCA, surgical procedures such as carotid-axillary, carotid-subclavian, aorta-subclavian, and axilla-axillary bypasses, as well as transposition of the internal mammary artery could be done [15,16]. These are the only option for revascularization despite the relatively high risk of procedures, as Carotid-subclavian bypass has a mortality rate of 0% at 30 days, 82% symptom-free survival rate at 5 years and 92% and 95 % primary and secondary patency rate at 10 years [16,17]. Indications for surgical bypass procedure include complete occlusion near the ostium of the vertebral artery, severe calcification of lesion with a length more than 5 cm, and concomitant brachiocephalic and coronary artery disease [15,18]. Peripheral stenting was preferred over carotid subclavian artery bypass in our patient as it is more feasible, minimally invasive and technically less difficult.

Conclusion

In post-CABG patients presenting with recurrent episodes of angina, a differential of coronary steal syndrome should always be kept in mind other than native coronary artery or graft vessel occlusion. History followed by a thorough physical examination is generally sufficient to diagnose CSSS. However, due to the heavy workload of OPD patients, clinical examination is generally overlooked leading to diagnostic dilemmas like in the present case. Moreover, LIMA ostium and subclavian artery ostium must always be profiled before referring patients for CABG and we should always look for pull back gradient while withdrawing the catheter in our case, this was the only clue that helped us reach the diagnosis. CT Coronary angiography if done in post-CABG patients should also include the ostial part of grafts as well as the subclavian artery to rule out significant stenosis. Such patients once diagnosed can be managed with peripheral stenting as in our case.

Statements and Declarations

Conflicts of interest

The authors declares that they do not have conflict of interest.

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Authors Contributions

All the authors have contributed equally to the work and fulfil ICMJE authorship criteria. All the authors have reviewed the final draft and approved the same.

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