

ANGINA PECTORIS ASSOCIATED WITH CORONARY-SUBCLAVIAN STEAL SYNDROME: CASE REPORT

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ABSTRACT

Introduction: Coronary-subclavian steal syndrome (CSSS) is a rare cause of myocardial ischemia after myocardial revascularization surgery (0.1% to 6%), with the main etiology being atherosclerosis. CSSS has an incidence of 3% in the population with atherosclerotic disease. The existence of peripheral vascular disease is the best predictive factor for the occurrence of CSSS. Prevalence is difficult to determine as many patients do not experience symptoms due to the development of an additional collateral network. Treatment is centered on correcting subclavian artery stenosis. Over the last decades, endovascular revascularization of subclavian arteries has presented excellent technical success rates (97%) and patency (5-year patency rate of 89–95%), comparable to surgical revascularization, with the advantage of being a minimally invasive technique, with morbidity and mortality rates (4.5%) lower than surgery and associated with shorter hospitalization, as well as faster recovery. We report a case of angina pectoris, in a post-CABG patient, with SRCS as the etiology and how the diagnosis and management of the case were made.

Keywords: Angina pectoris, Myocardial Revascularization, Subclavian Artery.

INTRODUCTION

The coronary-subclavian steal syndrome (CSSS) was first described in 1974 by Harjola and Valle. In patients undergoing coronary artery bypass grafting (CABG) using internal mammary arteries (IMA) as conduits, the presence of stenosis in the subclavian arteries proximal to the origin of the IMA causes a decrease in blood flow to the upper limb, leading to the reversal of flow in the IMA, with concomitant hemodynamic “steal” from the coronary circulation to the upper limb.¹

According to Vieira et al.², CSSS is a rare cause of myocardial ischemia after CABG (0.1% to

6%), with atherosclerosis being the main etiology. Cases have also been described in patients with Takayasu arteritis or left internal mammary artery malformations, such as arteriovenous fistulas. CSSS has an incidence of 3% in the population with atherosclerotic disease. The presence of peripheral vascular disease is the best predictive factor for the occurrence of CSSS. The prevalence is difficult to determine, as many patients are asymptomatic due to the development of a collateral network.³ The syndrome usually emerges between two and 31 years after CABG (mean age of 14 years), indicating that occlusive lesions developed after the mammary graft, with the onset of CSSS within one year after CABG suggesting that the stenosis of the left subclavian artery (LSA) was not detected at the time of cardiac surgery.⁴

Patients may be asymptomatic; however, the diagnosis should be considered in those undergoing CABG using the left internal mammary artery (LIMA) who present with cardiac symptoms such as angina-like chest pain and episodes of arrhythmia, as well as non-cardiac symptoms such as dizziness, vertigo, ataxia, and upper limb claudication. The condition is usually triggered or exacerbated by physical exertion. The physical examination should look for supraclavicular murmurs, pulse asymmetry, and, most importantly, a difference in blood pressure (BP) between the upper limbs >20 mmHg, with the latter being the most significant finding. Color Doppler ultrasound is a valid test for detecting hemodynamically significant stenosis in the subclavian territory, and images from computed tomography angiography (CTA) and magnetic resonance angiography (MRA) can also be considered for this purpose. However, digital subtraction angiography remains the gold standard for diagnosis. In this method, after contrast injection into the left anterior descending artery, the reverse flow of the LIMA toward the subclavian bed can be observed.⁴

The treatment is centered on correcting the subclavian artery stenosis. Previously, the recommended treatment was surgical and involved reimplanting the internal mammary artery (IMA) into the aorta to ensure graft patency and treat the subclavian stenosis, through carotid-subclavian grafting, aorto-subclavian grafting, or subclavian-carotid transposition. Although these procedures have high success rates and good long-term results, surgery is labor-intensive and subject to complications. Endovascular revascularization of the subclavian arteries has shown excellent technical success rates (97%) and patency rates (5-year patency rate of 89-95%) over the past decades, comparable to those of surgical revascularization. The advantage of endovascular treatment is that it is minimally invasive, with lower morbidity and mortality rates (4.5%) compared to surgery, as well as shorter hospital stays and faster recovery.¹

Our objective with this case report is to raise awareness of the possibility of this disease in patients undergoing CABG with episodes of angina, given its rarity and low prevalence.

CASE REPORT

An 85-year-old male patient presented to the emergency department (ED) four days before the consultation with a recent onset of edema in the lower limbs (LL) associated with episodes of precordial burning chest pain of mild intensity, occurring at rest and with upper limb mobilization, lasting less than five minutes and resolving spontaneously, starting earlier that week. He reports having had episodes of blood pressure differences in the upper limbs for the past two months. No pain was present at the time of the consultation. On examination, the patient appeared in good general condition, with a normal complexion and

hydration status. Blood pressure in the right upper limb (RUL) was 180/100 mmHg, and in the left upper limb (LUL) was 150/85 mmHg. Cardiac auscultation: regular rhythm, no murmurs, normal heart sounds, presence of a murmur ++/6 at the right subclavian site, edema in the lower limbs (LL) ++/4+.

The patient has a medical history of CABG in 2005 (left mammary artery to marginal branch 2, right mammary artery to left anterior descending artery), hypothyroidism, systemic arterial hypertension, mixed dyslipidemia, non-specific interstitial pneumonia, and is a former smoker. No known drug allergies. He is on continuous use of Syntroid® 150mcg, Livalo® 4mg, Ezetimibe 10mg, Concor® 5mg, Benicar anlo® 20/5mg, Clopidogrel 75mg, Dexilant® 30mg, Addera® 10,000, and Lipless® 100mg.

The patient underwent complementary exams in the emergency department: Chest X-ray showed bilateral opacities with an interstitial component. Electrocardiogram revealed sinus rhythm, within normal limits. Laboratory results showed BNP 99 pg/ml; Troponin 7.5 and 7.4 ng/ml. An angioCT (Figure 1) of the aorta and central vessels was requested during the consultation, which demonstrated: no aneurysm, dissection, mural hematoma, or ulceration. A predominantly calcified plaque was observed in the proximal portion of the right subclavian artery, extending approximately 0.8 cm, causing severe stenosis and occupying nearly the entire lumen in this area. In the other segments assessed, the subclavian arteries showed signs of mild atheromatosis without hemodynamically significant stenosis, including along the thoracic outlet bilaterally.

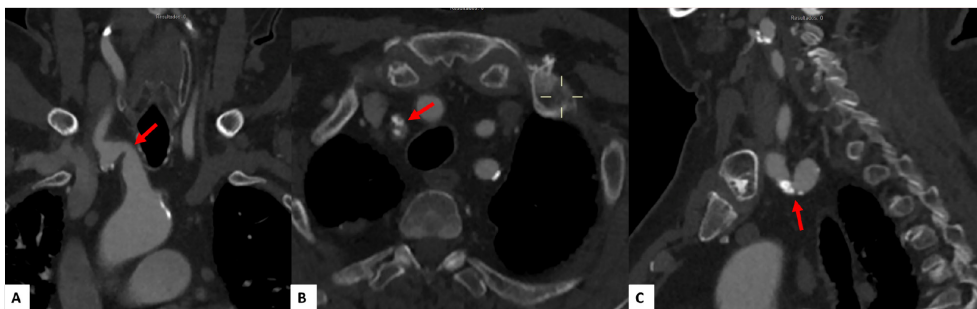


Figure 1: Angiotomography. The red arrow indicates stenosis in the right subclavian artery near the bifurcation of the brachiocephalic trunk.

After discussion with the radiologist and vascular surgeon, it was decided to perform a catheterization for better evaluation of the coronary grafts. The findings were as follows: the right coronary artery showed 50% stenosis at the mid-proximal transition and 30% stenosis in the distal third. The trunk had 50% stenosis in the distal third. The left anterior descending artery (LAD) had ostial occlusion. The right mammary artery to LAD graft was patent and functioning with a good anastomosis. The circumflex artery had a 90% calcified stenosis at its origin. The left mammary artery to marginal branch 2 graft was patent and functioning with a good anastomosis. The access route showed diffuse atherosclerosis and significant stenosis along the path, identifying a subocclusive stenosis of the right subclavian artery.

After review of the exam, it was decided to proceed with angioplasty in the stenotic region, using a right brachial artery puncture after dissection. A 12x40mm stent was implanted with significant technical difficulty due to tortuosity, but it was successfully placed in the proper position after using a centimeter-sized pigtail catheter to assess diameters. However, the stent migrated proximally due to the force of the stenosis, without distal embolization. A new puncture was necessary through the right femoral artery, where the guidewire was advanced, and a new 10x40mm stent was implanted in the correct position using the “wire mesh” technique, followed by balloon dilatation with a 7x40mm balloon catheter. Control angiography showed correction of the stenosis and proper stent placement, with no signs of dissection or embolization (Figure 2).



Figure 2: Angiograms. A: Pre-angioplasty image showing subclavian stenosis. B: Post-angioplasty image showing stenosis with stent.

DISCUSSION

SCVS is defined as the reversed blood flow from a coronary artery through an internal mammary artery graft toward the mid-distal subclavian artery, and occurs due to significant stenosis or total occlusion of the proximal portion of the latter. It is a rare, yet significant, cause of cardiac ischemia after coronary artery bypass surgery.⁴ This report discusses the most commonly used treatment for this complication today: percutaneous transluminal angioplasty to correct the affected subclavian stenosis.

Since the 1990s, percutaneous transluminal angioplasty has been considered the effective treatment for subclavian artery stenosis (SAS). The technique, followed by stent implantation, provides more anatomical and physiological results when compared to open surgery, and is associated with low morbidity, zero mortality, and short hospital stays. The short-term technical success rate is >90%, and long-term follow-up shows patency rates greater than 90% at five years.⁴ One of the largest studies, including 170 patients who underwent stent placement in subclavian or innominate arteries, reported a technical success rate of 98.3%, with 99.4% for stenotic lesions and 90.5% for occlusions. There were no procedure-related

deaths, and a stroke occurred in 0.6% of cases. In long-term follow-up, 82% of all treated patients remained asymptomatic, with a primary patency of 83% and secondary patency of 96%.⁵

The “clothesline” technique used for the correction of aortic aneurysms involves passing a guidewire from the femoral artery to the brachial artery and then pulling its ends, straightening the artery through a stretching mechanism. This allows for better passage of the delivery system for the endoprosthesis and facilitates the subsequent placement of the stent.⁶ This technique was employed in the procedure in question due to anatomical difficulty and the unsuccessful implantation of the first stent using the conventional technique.

The occurrence of restenosis after endovascular procedures is relatively low, around 16% at 5 years, and can be treated with repeat procedures. Thus, endovascular revascularization is now used as the first-line technique for treating SRCS. Surgical revascularization remains an important technique, used when it is not possible to bypass the lesion during angioplasty, in calcified occlusive lesions, in long obstructive lesions, and in certain cases of restenosis.¹

In conclusion, we would like to consolidate the diagnostic hypothesis of subclavian-coronary steal syndrome as a differential diagnosis for angina, highlighting endovascular procedures as a first-line therapeutic option, with high short-term success and long-term patency.

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