CORONARY-SUBCLAVIAN STEAL: CASE SERIES AND REVIEW OF THE LITERATURE

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Abstract

Coronary subclavian steal syndrome (CSSS) is a relatively uncommon entity, and its clinical spectrum is characterized by stable exertional angina and rarely as acute coronary syndrome. The diagnosis can be established easily by angiography.

We report a case series of three patients with CSSS and acute coronary syndrome and we review the literature in the attempt to understand the nature of symptomatology and the mechanisms of ischemia in this condition. Our study raised some questions about the correct definition of this entity, the pathophysiology of coronary steal and the mechanisms of ischemia, in the setting of unstable angina and acute myocardial infarction.

Keywords: coronary artery bypass, left internal mammary artery, subclavian artery stenosis, coronary subclavian steal syndrome, acute coronary syndrome.

Introduction

The use of the left internal mammary artery (LIMA) is associated with the best long-term patency of coronary artery bypass surgery, because this conduit is spared from atherosclerosis. However, this is not true for the proximal ("inflow") part of this "ideal" conduit, the subclavian artery, where atherosclerotic stenosis can compromise the vessel.

We describe the cases of 3 patients with coronary subclavian steal syndrome (CSSS) with the following clinical manifestations: two with resting angina and one with acute myocardial infarction.

Case 1

A 58 year-old man was admitted to the hospital with intermittent spontaneous anterior chest pain radiating to the left arm, dizziness and dyspnea. He had a history of coronary artery bypass grafting performed 6 years earlier [LIMA to LAD (left anterior descending artery) and SVG (safenous vein graft) to RCA (right coronary artery)], followed by percutaneous angioplasty and stenting of the circumflex artery 2 years afterwards. The physical examination on admission revealed the presence of pulse in the right radial artery and absence in the left. ECG (electrocardiography) demonstrated anterior ischemia during the chest pain episodes. Echocardiogram showed a LVEF (left ventricular ejection fraction) of 39% (unchanged from previous examinations). Laboratory tests revealed normal cardiac enzymes. The Doppler study highlighted significant left subclavian artery stenosis with vertebral subclavian steal pathophysiology and a non-stenotic ipsilateral carotid artery. Coronary angiography revealed the patency of the two bypass grafts and of the native circumflex artery.

Aortography demonstrated severe (90%) stenosis of the proximal left subclavian artery - which was felt to be responsible for the above mentioned symptoms – while the vertebral artery was not visualized. The lesion was stented with an autoexpandable stent (SelfX 8/44 mm, Abbott Medical Devices, Beringen, Switzerland), after pre-dilation. An excellent angiographic result was obtained after post-dilation with a Fox Plus balloon of 9/40 mm, Abbott Laboratories Vascular Enterprises ltd., Switzerland (Fig. 1 A, B).
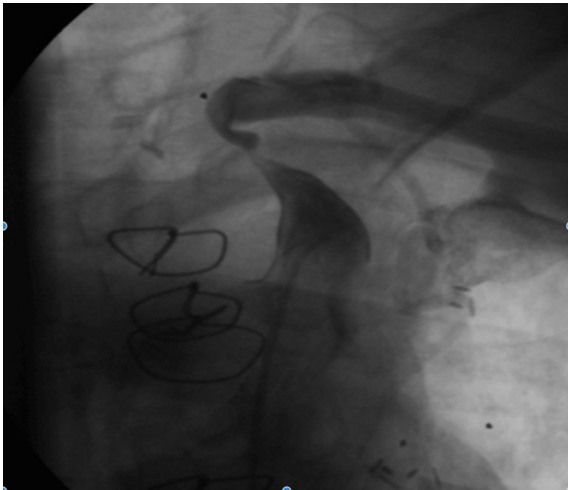
Figure 1A. Severe subclavian artery stenosis before stenting.

Figure 1B. After stenting, the flow through the vertebral artery can be seen.

Case 2

A 59 year-old man with diabetes mellitus, severe hypertension, old inferior myocardial infarction and peripheral arterial disease, had CABG (coronary artery bypass grafting) for unstable angina in 1998. The patient received LIMA to LAD and SVG to the RCA. In 2007 he presented again with resting angina and anterior wall ischemia on the ECG, but with no elevation in cardiac biomarkers. LVEF was 42% by echocardiography, and both clinical examination and Doppler study evidenced a severe left subclavian artery stenosis, along with 70% bilateral internal carotid artery stenosis. On the angiogram, the saphenous vein graft to the right coronary artery was found to be occluded, and the severe stenosis of the left subclavian artery was confirmed. The subclavian artery stenosis was stented with Palmaz Genesis 7/24 mm, Cordis Europe N.V., Roden, Netherlands, and expanded with an 8/20mm Fox Plus balloon, Abbott Laboratories Vascular

Enterprises ltd., Switzerland.

Three months later, because of exertional angina with a positive exercise stress test, we performed stenting of the right coronary artery. Two 3.5/23 Bx Sonic stents (Cordis, Johnson & Johnson, Warren, New. Jersey) were implanted in the right coronary artery and were expanded at 18 atm, with good angiographic results.

Six months later, the patient returned for resting angina, with anterior ischemic ECG changes during pain, and normal troponin. The coronarography showed a high degree in-stent restenosis in the subclavian artery. Redilation was performed with a 8/20mm Fox Plus balloon, Abbott Laboratories Vascular Enterprises ltd., Switzerland at 16 atm, until the residual stenosis was minimal and the pressure gradient became zero. (Fig. 2 A, B, C, D).
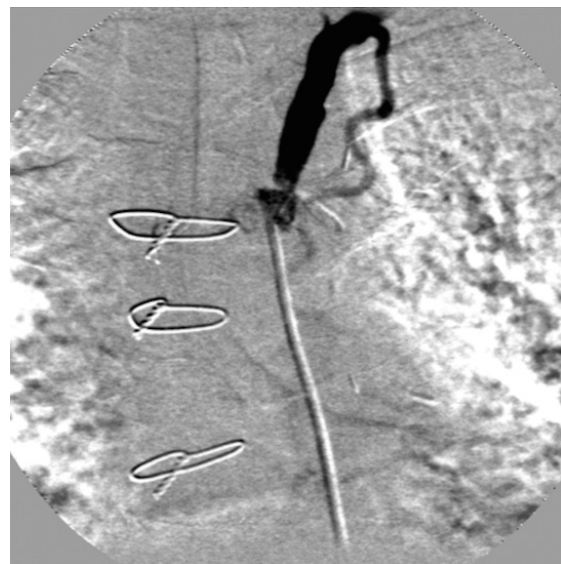

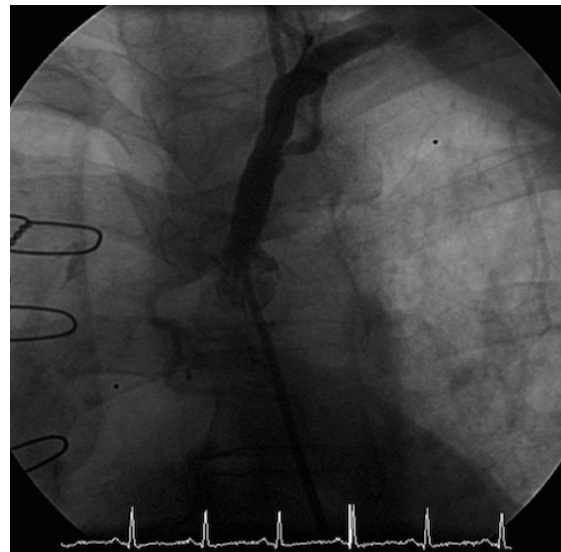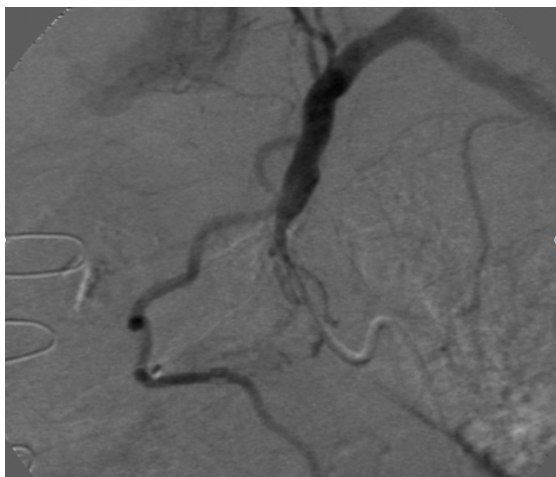**Figure 2A.** Tight ostial subclavian artery stenosis.

Figure 2B. Subclavian artery after stenting.

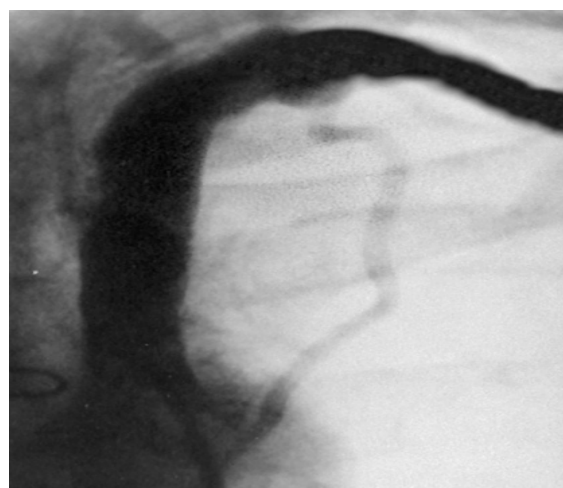**Figure 2C.** In-stent restenosis of the left subclavian artery.

Figure 2D. The vessel after balloon angioplasty, no residual stenosis is seen.

Figure 3A. Severe stenosis of left subclavian artery.

Figure 3B. The subclavian artery after stenting.

Case 3

A 51 year-old man presented in 2001 with chest pain at rest and dyspnea. He had a history of prior inferior myocardial infarction (1996) and had CABG few months later (LIMA to LAD and SVG to the RCA). At presentation, the ECG revealed inferior wall ischemia as well as 2 mm transient ST segment elevation in V1-V4. The CK-MB (creatine kinase myocardial band fraction) was increased. Echocardiogram showed an EF (ejection fraction) of 45%. Coronary angiography, performed after 24 hours after admission, demonstrated patency of the two grafts, proximal high degree LAD stenosis and severe stenosis of the proximal left subclavian artery - which was considered the culprit lesion. This was stented using a Jostent 4-9 mm/28 mm (Jomed GmbH, Rangendingen, Germany), mounted on a 9x4 cm OPTA balloon angioplasty catheter (Cordis, Miami Lakes, FL). No residual stenosis was noted and the clinical status significantly improved (Fig. 3 A, B).

Discussion

Some considerations need to be made regarding the above mentioned cases of "acute coronary syndrome". Coronary-subclavian steal syndrome is defined as a coronary reversal of flow in the internal mammary artery caused by proximal subclavian artery stenosis, producing myocardial ischemia [1,2,3] (Fig. 4).

In this setting, the cardiac symptoms could be stable exertion angina, silent ischemia or, less frequently, angina with acute coronary syndrome. After the review of the literature and our case series particularities, some questions arise.

Our concerns is the existence of unstable angina in the setting of CSSS. Could this possibly be an acute coronary syndrome, or is it a special form of unstable angina, unrelated to any underlying atherosclerotic unstable plaque?

However, literature also describes cases of CSSS where enzymatic alterations suggested an acute myocardial infarction, as has been in our case too (case no. 3), even

Figure 4. Left coronary coronarography reveals late opacification of the stenosed subclavian artery by LIMA reflow.

if this scenario is considered rare [1,2,3,4,5]. One possible mechanism involves plaque destabilization in the native coronary or subclavian artery, with distal microembolization from the affected plaque. But it is hard to believe that years after acute coronary syndrome, the culprit LAD lesion remained unstable for such a long time. However this is not totally impossible, as CABG does not stabilize the plaque, but rather eliminates the hemodynamic consequences of a tight stenosis. Another mechanism which could explain this issue is a new unstable stenosis on the LAD, in the hemodynamic condition of CSSS. The natural history of unstable lesions can go with spontaneous healing in up to one year [6,7]. A second possible mechanism - the unstable plaque stenosis in the subclavian artery that would only embolize into the LIMA - is hard to accept, because the steal that directs the bloodstream from LIMA to the subclavian artery would also lead to embolization in the digital artery [3,8].

Should thus the diagnosis of acute coronary syndrome be redefined in this condition of CSSS, and the term unstable angina secondary to the decrease of coronary flow and coronary steal be accepted instead? Similarly, should we reconsider our acute myocardial infarction (AMI) cases as, in fact, AMI type 2 cases, or do we accept the existence of microembolism of unclear origin (left main, LAD or subclavian artery) instead? In case of stenosis or occlusion of the subclavian artery, coronary flow in the LAD territory depends on the following factors: the flow from the native vessel (if still existent), the flow through the internal mammary artery (which depends to a great extent on the peripheral vascular resistance of the subclavian artery and retrograde flow from the vertebral), and the collateral flow from other coronary areas, if present.

Probably the low flow in the above settings could explain a Type 2 myocardial infarction.

Conclusions

The clinical spectrum of CSSS includes exercise angina, silent ischemia or even AMI with or without ST segment elevation, depending on local hemodynamics and not only. The relation between anterograde flow through the native subclavian artery (depending also on the vertebral steal) and the coronary collaterals, as well as the peripheral vascular resistance in the subclavian artery, dictates the degree of myocardial ischemia.

Acute myocardial infarction in the setting of CSSS is most likely due to a type 2 mechanism, as per the most recent AMI classification [9], and not to an ulcerated or complicated atherosclerotic plaque. However this issue of unstable atherosclerotic plaques could not be completely neglected, as a complementary mechanism of ischemia in these cases. Thinking so, even if we perform the subclavian revascularization which involves aggressive double anti-aggregant therapy, there is need of high doses of atorvastatin and ACE (angiotensin-converting-enzyme) inhibitors for their pleiotropic effect.

References:
1. Lelek M, Bochenek T, Drzewiecki J, Trusz-Gluza M. Unstable angina as a result of coronary-subclavian steal syndrome. Circ Cardiovasc Interv. 2008;1:82-84.
2. Pappy R, Kalapura T, Hennebry T.A. Anterolateral myocardial infarction induced by coronary-subclavian-vertebral steal syndrome successfully treated with stenting of the subclavian artery. J Invasive Cardiol. 2007;19(8):E242-245.
3. Takach T, Reul GJ, Cooley D, Duncan JM, Livesay JJ, Ott DA, et al. Myocardial thievery: the coronary-subclavian steal syndrome. Ann Thorac Surg. 2006;81(1):386-392.
4. Tan JW, Johan BA, Cheah FK, Wong P. Coronary subclavian steal syndrome: a rare cause of acute myocardial infarction. Singapore Med J. 2007;48(1):5-8.
5. Costa SM, Fitzsimmons PJ, Terry E, Scott RC. Coronary-subclavian steal: case series and review of diagnostic and therapeutic strategies: three case reports. Angiology. 2007;58(2):242-248.
6. Forrester JS. Toward understanding the evolution of plaque rupture: Correlating vascular pathology with clinical outcomes. J Am Coll Cardiol. 2003; 42:1566-1568.
7. Rioufol G, Gilard M, Finet G, Ginon I, Boschat J, André-Fouët X. Evolution of spontaneous atherosclerotic plaque rupture with medical therapy: long-term follow-up with intravascular ultrasound. Circulation. 2004;110:2875-2880.
8. Blumenthal RS, Savader SJ, Resar JR, Brinker JA, Reis SE. Use of intravascular Doppler ultrasonography to assess the hemodynamic significance of the coronary-subclavian steal syndrome. Am Heart J. 1995;129(3):622-625.
9. Thygesen K, Alpert JS, White HD, Joint ESC/ACCF/AHA/WHF Task Force for the Redefinition of Myocardial Infarction. Universal definition of myocardial infarction. J Am Coll Cardiol. 2007;50(22):2173-2195.