



Coronary Subclavian Syndrome Case Series and Review of Therapeutic Aspects

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Abstract

Background: Coronary-subclavian steal syndrome (CSSS) is a clinical condition characterized by the reversal of blood flow in the left internal thoracic artery (ITA) in patients who have undergone coronary revascularization using this artery. It is a rare cause of myocardial ischemia subsequent to stenosis or occlusion of the subclavian artery (SA) proximal to ITA coronary bypass. Proximal subclavian artery (SA) stenosis is present and atherosclerotic disease is the underlying pathophysiologic mechanism in the majority of cases. We report a series of patients with late CSSS treated through an endovascular approach.

Keywords:

Coronary-subclavian steal syndrome; Coronary bypass; Internal mammary artery; Subclavian stenosis; Myocardial ischemia.

Introduction

The coronary-subclavian steal syndrome (CSSS), first described in 1974 by Harjola and Valle, develops in 0.5% to 4% of patients undergoing myocardial revascularization (MR). Usually refers to a reversed blood flow from the myocardium to the upper limb due to ipsilateral subclavian artery stenosis (SAS) in patients with previous revascularization using the internal thoracic artery (ITA).

The use of left ITA for coronary artery revascularization has been associated with better long-term patency and patient survival than the use of a saphenous venous graft because in situ has superior patency rate and survival benefit when

grafted to the left anterior descending artery. It is well known that each subclavian artery (SA) has four main branches, the ITA being one of them. The use of the ITA is better associated with long-term MR surgery due to being a conduit usually spared from atherosclerosis. However, this is not true for the proximal (“in flux”) part of this conduit, the subclavian artery, where atherosclerotic stenosis can affect the vessel. Atherosclerosis represents more than 90% of subclavian artery stenosis. Less common etiologies include arteritis (for example, Takayasu’s arteritis, giant cell arteritis), inflammation, radiation exposure, compression syndromes, fibromuscular dysplasia, and neurofibromatosis.

CSSS should be suspected in patients undergoing revascularization using the ITA with different pulse and blood pressure in the upper limbs, and clinical presentations of angina. Significant stenosis or even occlusion of the subclavian artery proximal to the ITA can lead to decreased blood flow to the upper limb, triggering a flow inversion in the ITA with a concomitant hemodynamic “steal” of coronary blood flow to the upper limb. CSSS patients usually present stable or unstable angina and may also present arm claudication and several neurological symptoms⁸⁻¹⁰. CSSS consequences can include ischemic cardiomyopathy, acute myocardial infarction (AMI), cerebrovascular accident, and death. CSSS is treated by correcting the stenosis or occlusion of the subclavian artery.

This study describes the cases of four patients with CSSS with the following clinical presentations: three with stable angina and one with unstable angina.

In addition, a review of the therapeutic aspects is provided.

Methods

The clinical data of 4 consecutive patients with CSSS who had undergone subclavian artery stenting between 2015 and 2019 were reviewed. The anatomic and clinical-angiographic characteristics of the series were considered. Follow-up was performed and a review of the therapeutic aspects is provided.

Results

General characteristics

From January 2015 to December 2019, 4 CSSS patients were treated; 3 with stable angina and 1 with unstable angina. Of the 4 patients, 3 had subclavian artery stenosis (2 ostial at the origin and 1 in the middle segment), and 1 had proximal occlusion of the left subclavian artery. The clinical-angiographic characteristics of the patients are described. The access of choice was the brachial artery. In 1 case of proximal occlusion of the left subclavian artery, simultaneous femoral and percutaneous brachial access was necessary. All cases underwent pre-dilation of the stenotic lesion. Balloon-expandable stents were used in all patients with stenosis or proximal ostial occlusion, and a self-expanding stent was used in 1 case with non-ostial lesion in the left subclavian artery. No patient developed angina during the follow-up period (12+4 months). The patient with unstable angina had total ostial occlusion of the left subclavian artery. Technical

success was achieved in all 4 patients. There were no complications at the puncture site and no clinical cardiac or neurological presentations developed during surgery or in the immediate postoperative period. No significant hematoma was observed postoperatively. All procedures were performed with ultrasound-guided puncture. At a mean follow-up of 12+4 months, all patients were asymptomatic for new anginal symptoms, without indirect signs of restenosis on duplex ultrasound performed on the limb.

Conclusion

CSSS is a serious and silent pathological entity resulting from the extensive atherosclerotic involvement of the left subclavian artery. The clinical spectrum of the syndrome includes silent ischemia, anginal symptoms, or even AMI, depending on the local hemodynamics of the lesion. Its prevalence is underestimated and duplex ultrasound, computed tomography, or angiography of the subclavian artery should be considered in all patients with suggestive clinical findings. Restoring the anterograde ITA flow is the key to preserve coronary perfusion. Currently, the endovascular approach is the technique of choice due to its high success rates, low morbidity, and mortality and for being minimally invasive.

Biography

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