Percutaneous Treatment of Refractory Angina Secondary to Left Subclavian Artery Stenosis in Patients after Coronary Artery Bypass Grafting

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Introduction

Anginal chest pain is usually related to atherosclerotic coronary artery disease and its clinical presentation differs depending on the pathological aspects of the lesions.

Left subclavian artery stenosis (SAS) as a cause of angina pectoris (AP) is often reported in patients with a history of coronary artery bypass grafting through left internal mammary artery (LMA) involved in the process of subclavian stenosis1. Very often, this diagnosis may be overlooked especially when patients are asymptomatic and present ischemic abnormalities on scintigraphy. Subclavian stenosis may also be associated with neurological symptoms by virtue of reversal of the flow through the vertebral artery due to subclavian hypotension.

This is the case of a patient who underwent coronary artery bypass grafting with LMA for anterior descending artery (ADA) not involved in the subclavian stenosis and typical growing anginal pain related to physical exertion with the upper limbs or predominant involvement of the limbs. Stent-assisted percutaneous correction of the ostial and subtotal subocclusion of the left subclavian artery (LSA) made the patient asymptomatic.

Case Report

Male patient, 72 years old, taxi driver, type 2 diabetes, former smoker with a history of coronary artery bypass surgery four years before. The patient was asymptomatic four months before. Then, anginal pain on exertion that rushed or worsened when the patient used the upper limbs associated with frequent tiredness in the left arm. The anginal pain acquired characteristics of instability over time, and the patient was sent to an emergent coronary angiography with angina at rest.

The selective injection of contrast into the left coronary artery opacified the left subclavian artery via retrograde flow of contrast through the ipsilateral mammary artery and slow flow with slight distal opacification of the anterior descending branch after anastomosis.

The injection of contrast in the proximal third of the left subclavian artery or in the transverse aorta showed ulcerated and subtotal lesion of the LSA ostium with slight flow (Figure 1). The absence of opacification of the left internal mammary artery was a source of concern.

After stenting in the LSA ostium, the flow of contrast was restored, as well as the anatomy of the segment (Figure 2). Normal flow of contrast through the left internal mammary artery was observed, completely opacifying the distal bed of the anterior descending artery. A new injection of contrast into the native left coronary trunk showed the absence of retrograde flow through the LMA towards the LSA.

Discussion

SAS is relatively frequent and, in general, it is an atherosclerotic condition that is more common in diabetics, smokers and in those suffering from peripheral arterial disease. The correlation of cerebral vascular accident and coronary artery disease with SAS has received little attention2. However, Reivich et al reported, in 1961, two patients with clinical signs of cerebrovascular insufficiency associated with reversal of flow through the vertebral artery, secondary to subclavian obstruction. The term “subclavian steal syndrome” was introduced by Fisher in 1962. Since then, a few cases have been reported in the literature4, because of its low incidence in the general population. Another anatomic aspect that also characterizes a flow steal syndrome in these patients occurs when the intercostal branches of great importance originate from the left mammary artery and do not suffer ligation during the coronary artery bypass surgery.

Symptoms of SAS in the arms are claudication, reported by the patient, digital cyanosis and, in severe cases, distal necrosis by embolism. However, the most striking but rare symptoms are those affecting the brain and heart. Neurological symptoms in general characterize vertebrobasilar syndrome and the most common condition is vertigo. Symptoms of myocardial ischemia are generally found in patients that underwent coronary artery bypass grafting when there is involvement of the internal mammary artery in the SAS process. In our case, although no involvement of the mammary artery was observed in the SAS, the distal coronary flow in the left anterior descending artery supplied by the mammary artery was impaired due to diversion of blood flow to the left subclavian artery5,6.

Keywords

Subclavian Steal Syndrome; Myocardial Revascularization; Angioplasty, Balloon, Coronary.
Figure 1 – Aortography with digital subtraction and selective angiography of left subclavian artery displaying subtotal and ostial stenosis of LSA, as well as absence of opacification of LMA.

Figure 2 - Selective angiography with digital subtraction of LSA after successful stenting. Observe normal opacification of LMA.
Thus, the increase in oxygen consumption determined by exertion in the left upper limb stimulated retrograde flow through the mammary artery, thus reducing distal perfusion of the left anterior descending artery. Hence the concomitant angina and claudication of the left arm.

Regarding the SAS, the indirect measurement of blood pressure in both arms is paramount and essential for the clinical suspicion. The difference in systolic blood pressure between the upper limbs above 15 mmHg correlates well with positive angiography, the gold standard for diagnosing SAS. This is true even though the sensitivity is quite lower than specificity. This fact is generally attributed to cases of bilateral involvement or non-significant unilateral stenosis. Since most patients with SAS are asymptomatic, the measurement of blood pressure with cuff on one arm only may incorrectly determine blood pressure, not controlling a potential hypertension and failing to raise an important diagnostic suspicion. In this case, the finding of blood pressure difference was only found after angiographic diagnosis given the urgency of the case.

Our patient is a taxi driver, age 72, and was referred for emergency coronary angiography with clinical picture of persistent angina at rest.

As risk factors for SAS, the patient presented type II diabetes mellitus using insulin, history of smoking for 50 years and dyslipidemia. Four years before, he had underwent coronary artery bypass surgery with placement of two bypass grafts and anastomosis of the left internal mammary artery to the anterior descending artery in the middle third. Four months before, the patient was asymptomatic, although with an irregular clinical control.

In his clinical history, there was onset or worsening of anginal chest pain at exertion involving the upper limbs, mainly the left ones. His left upper limbs were easily fatigued.

The selective injection of contrast into the left coronary trunk showed opacification of the left subclavian artery retrogradely through the left internal mammary artery with reduced distal flow to the anterior descending artery. There was no common “flow competition aspect” between the LMA and the proximal ADA.

The injection of contrast into the proximal third of the LSA and the transverse aorta revealed ostial subocclusion of that branch with almost no flow through it. No LMA opacification was attributed to the reverse flow.

The patient was immediately referred to successful and uneventful balloon angioplasty followed by stenting (6.0 x 12 mm Palmaz Genesis – Cordis, USA) in the stenosed segment.

During immediate eight-week follow-up, the patient remained asymptomatic even when subjected to exertion involving the upper limbs, which are now with equalized blood pressure.

**Author contributions**

Conception and design of the research, Acquisition of data, Analysis and interpretation of the data, Writing of the manuscript: Osterne EMC, Osterne ECV, Motta VP, Motta PAM, Salame CK, Osterne TEC; Statistical analysis, Obtaining funding and Critical revision of the manuscript for intellectual content: Osterne EMC, Osterne ECV, Osterne TEC.

**Potential Conflict of Interest**

No potential conflict of interest relevant to this article was reported.

**Sources of Funding**

There were no external funding sources for this study.

**Study Association**

This study is not associated with any post-graduation program.

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