

Case Report

Surgical Treatment of Coronary Subclavian Steal Syndrome

MIHALIS ARGIRIOU¹, VASSILIOS FILLIAS¹, DIMITRIOS EXARHOS², VICTOR PANAGIOTAKOPOULOS¹
ILIAS KOUERINIS¹, CHARALAMBOS ZISIS³, ANTONIA DIMAKOPOULOU⁴, ION BELLENIS³

¹Cardiac Surgery Department, ²Radiology Department, ³Thoracic and Vascular Surgery Department,
⁴Anesthesiology Department "Evangelismos" General Hospital, Athens, Greece

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The internal mammary artery is the conduit of choice for cardiac revascularization. Atherosclerotic disease of the coronary arteries may simultaneously involve the subclavian artery. Proximal stenosis in the left subclavian artery may result in recurrent myocardial ischemia in patients with a patent left internal mammary artery (LIMA), due to coronary steal syndrome through the LIMA.

The use of the left internal mammary artery (LIMA) as a conduit to the anterior descending artery is recommended by the American College of Cardiology/American Heart Association as a coronary bypass graft¹ because of enhanced long-term survival with a well-documented long-term patency rate.²

The LIMA is rarely the source of atherosclerotic disease: however, there is a risk of ischemia of the myocardium supplied by the LIMA, if there is hemodynamically significant stenosis of the left subclavian artery, causing reversal of blood flow through the LIMA. This phenomenon is known as the coronary-subclavian steal syndrome (CSSS) and its incidence after coronary artery bypass grafting (CABG) is about 0.44%.

Case presentation

A 71-year-old man presented with chest pain, left arm claudication and rotational vertigo during mild physical activity related to his left upper extremity. Eleven years before he had undergone triple CABG (LIMA to left anterior descending artery and two saphenous vein grafts to the first

obtuse marginal and the right coronary arteries). Following surgery the chest pain resolved, but angina symptoms recurred ten years later.

Thorough physical examination, including bilateral simultaneous brachial blood pressure measurements, demonstrated a significant arterial pressure discrepancy between the upper extremities (right 125-60 mmHg, left 80-55 mmHg). A cerebral computed tomography scan was normal.

Duplex ultrasound scanning revealed proximal severe stenosis of the left subclavian artery with retrograde flow in the left vertebral artery. Both vertebral and carotid arteries were normal and patent, while a 20% stenosis of the right and a 30% stenosis of the left carotid artery bifurcation were detected without any significant compromise of the blood flow.

Coronary angiography revealed proximal subtotal occlusion of the left subclavian artery, patent venous grafts, and retrograde filling (steal) of the LIMA and the left subclavian artery during the injection of the contrast medium into the left coronary artery (Figure 1). Angiographic magnetic resonance imaging confirmed the subtotal occlusion of the left subclavian artery.

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Address:
Mihalis Argiriou

41 Aetidon St.
15561 Hologos
Athens, Greece
e-mail:
mihalisargiriou@ath.forthnet.gr

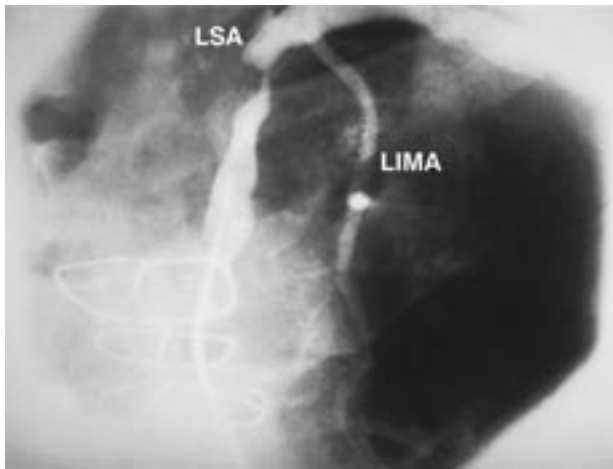


Figure 1. Coronary angiography demonstrating the proximal subtotal occlusion of the left subclavian artery (LSA). LIMA – left internal mammary artery.

The diagnosis of complete simultaneous coronary-subclavian and vertebral-subclavian steal syndrome was established according to the clinical, ultrasonographic, and angiographic findings.

Percutaneous transluminal angioplasty (PTA) to address the proximal left subclavian artery stenosis was attempted, but passage of the guide wire was not possible because of the chronic near-total occlusion.

A carotid to subclavian bypass by a transverse left supraclavicular incision was performed. An 8 mm diameter polytetrafluoroethylene (PTFE) graft was used. Postoperatively the patient remained under treatment with clopidogrel and aspirin (100 mg/day).

Three months after surgery, 16-row spiral computed tomographic angiography³ (CTA, Toshiba-Aguilion) of the aortic arch and branches was performed with simultaneous and continuous assessment of the coronary arteries by a cardiac computed tomography protocol. The CTA demonstrated a patent carotid-subclavian PTFE graft as well as normal opacification of the left anterior descending branch through the LIMA (Figures 2, 3).

The patient remains free of symptoms at 18-month follow-up with no divergence between blood pressure measurements in the two arms.

Discussion

Although early case reports of CSSS appeared in the literature in the 1970s, the syndrome is still considered an uncommon complication of myocardial

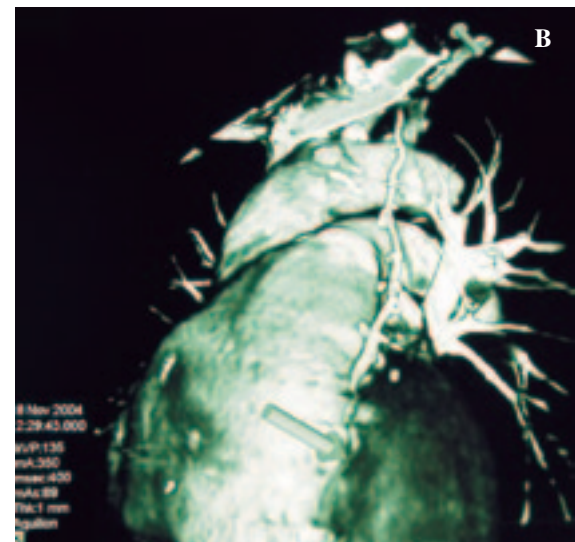


Figure 2. The 16-row spiral CT demonstrates (A) a patent carotid-subclavian polytetrafluoroethylene (PTFE) graft (white arrow), and (B) normal opacification of the left anterior descending branch through the left internal mammary artery (arrow).

revascularization. However, several authors have cautioned that its incidence may be higher than suspected because of a greater number of LIMA grafts and a longer life expectancy.

Athero-occlusive disease of the aortic arch after CABG, especially occlusion or severe stenosis of the subclavian artery proximal to the origin of the LIMA, may result in impaired myocardial blood flow. Usually, CSSS is associated with the onset of angina pectoris after stress of the upper limb and with symptoms related to posterior cerebral ischemia or upper ex-



Figure 3. The white arrow demonstrates the proximal subtotal occlusion of the left subclavian artery.

tremity ischemia. Occasionally, some patients remain asymptomatic or exhibit only silent ischemia.

Emergence of CSSS less than a year after coronary revascularization with an *in situ* internal mammary artery graft suggests that subclavian artery stenosis was probably missed at CABG. Usually CSSS occurs between 2 and 31 years after CABG (on average 14 years)⁴ indicating that the subclavian artery occlusive lesions most likely develop after internal mammary grafting. In our case, the patient developed CSSS 10 years after the CABG operation. No significant discrepancy in blood pressure between the right and the left arm nor any supraclavicular bruit was noticed at the routine preoperative physical examination.

Since 1980, carotid-subclavian artery bypass grafting was considered the procedure of choice for treatment of CSSS. The excellent patency rates of synthetic materials and its minimal operative risk have been documented by several reports.⁵⁻⁷ Since 1990, percutaneous transluminal angioplasty with stent implantation has been considered an effective treatment of subclavian artery stenosis. The technique is associated with a low incidence of morbidity and mortality, short hospitalization, excellent short-term and acceptable long-term results.⁸⁻¹⁰ However, restenosis after initially successful angioplasty remains a considerable problem, especially in certain groups of patients. In a recent study,¹¹ Ferrara et al reported that patients with complete CSSS present a higher risk of subclavian

restenosis (40.7% over 5 years). Schillinger et al¹² reported that subclavian arterial stent implantation is associated with better one-year patency than balloon angioplasty, due to improved technical success, but intermediate and long-term outcomes are less favorable, as in-stent restenosis occurs frequently (16%). Other studies have demonstrated a similarly high rate of recurrent stenosis, averaging 13% to 16%.^{8,13}

Moreover, patients with complete occlusion of the vessel and long or chronic lesions are less suitable for PTA, primarily because of problems with catheter passage through the lesion, despite the improvement in endovascular equipment and techniques over the last decade. De Vries et al¹⁴ reported a 100% technical success rate for stenosis and 65% for occlusions. In our case the patient developed symptoms ten years after the initial CABG procedure, while the coronary angiography and angiographic magnetic resonance imaging revealed proximal near-total occlusion of the left subclavian artery. It is possible that the extent and the chronicity of the lesion were responsible for the failure of the guide wire to cross the proximal part of the left subclavian artery.

When PTA is not feasible because of complete or chronic occlusion, extent of disease, stent failure, or when the LIMA perfuses more than one vessel, extra-anatomic surgical reconstruction provides a safe, effective, and durable treatment.

Although many authors advocate that PTA and stent placement should be considered as first line therapy for subclavian obstruction, there is still doubt concerning patients who have already had coronary revascularization with LIMA.¹⁵ Prospective, multicenter, randomized studies of larger populations are warranted to compare the short and long-term results of stent placement and surgical intervention in those patients.¹⁶

References

1. Guidelines and indications for coronary artery bypass graft surgery. A report of the American College of Cardiology/American Heart Association Task Force on Assessment of Diagnostic and Therapeutic Cardiovascular Procedures (Subcommittee on Coronary Bypass Graft Surgery). *J Am Coll Cardiol* 1991; 17: 543-589.
2. Cameron AA, Green GE, Brogno DA, et al: Internal thoracic artery grafts. *J Am Coll Cardiol* 1995; 25:188-192.
3. Nieman K, Pattynama PM, Rensing BJ, Van Geuns RJ, De Feyter PJ: Evaluation of patients after coronary artery bypass surgery: CT angiographic assessment of grafts and coronary arteries. *Radiology* 2003; 229: 749-756.
4. Westerband A, Rondriquez JA, Ramaiah VG, et al: Endovas-

- cular therapy in prevention and management of coronary-subclavian steal. *J Vasc Surg* 2003; 38: 699-704.
5. Ziomek S, Quinines-Baldrich WJ, Busutil RW, et al: The superiority of synthetic arterial grafts over autogenous veins in carotid-subclavian bypass. *J Vasc Surg* 1986; 3: 140-144.
 6. Aburahma AF, Robinson PA, Jennings TG: Carotid-subclavian bypass grafting with polytetrafluoroethylene grafts for symptomatic subclavian artery stenosis or occlusion: a 20-year experience. *J Vasc Surg* 2000; 32: 411-419.
 7. Paty PS, Mehta M, Darling RC, et al: Surgical treatment of coronary subclavian steal syndrome with carotid subclavian bypass. *Ann Vasc Surg* 2003; 17: 22-26.
 8. Milliare A, Trinca M, Marache P, et al: Subclavian angioplasty: immediate and late results in 50 patients. *Cathet Cardiovasc Diagn* 1993; 29: 8-17.
 9. Paraskevaidis SA, Giavroglou KE, Proios TD, et al: Stent implantation at subclavian artery in a patient with left internal mammary graft and subclavian steal syndrome. *Hellenic J Cardiol* 1997; 38: 310-315.
 10. Polydorou AA, Michalopoulos CD, Kouvaras GA, Vlachos LI: Coronary-subclavian syndrome: Report of a case treated with subclavian angioplasty. *Hellenic J Cardiol* 1993; 34: 643-648.
 11. Filippo F, Francesco M, Francesco R, et al: Percutaneous angioplasty and stenting of left subclavian artery lesions for the treatment of patients with concomitant vertebral and coronary subclavian steal syndrome. *Cardiovasc Intervent Radiol* 2006; 29: 348-353.
 12. Schillinger M, Haumer M, Schillinger S, et al: Risk stratification for subclavian artery angioplasty: is there an increased rate of restenosis after stent implantation? *J Endovasc Ther* 2001; 8: 550-557.
 13. Henry M, Amor M, Henry I, et al: Percutaneous transluminal angioplasty of the subclavian arteries. *J Endovasc Surg* 1999; 6: 33-41.
 14. De Vries JP, Jager LC, Van den Berg JC, et al: Durability of percutaneous transluminal angioplasty for obstructive lesions of proximal subclavian artery: long-term results. *J Vasc Surg* 2005; 41: 19-23.
 15. Peterson BG, Eskandari MK, Gleason TG, Morasch MD: Utility of left subclavian artery revascularization in association with endoluminal repair of acute and chronic thoracic aortic pathology. *J Vasc Surg* 2006; 43: 433-439.
 16. Ferrara F, Meli F, Raimondi F, et al: Subclavian stenosis/occlusion in patients with subclavian steal and previous bypass of internal mammary interventricular anterior artery: medical or surgical treatment? *Ann Vasc Surg* 2004; 18: 566-571.