

Positional Coronary Subclavian Steal Syndrome from Thoracic Outlet Compression

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Received: September 04, 2026; **Accepted:** September 22, 2026; **Published:** October 05, 2026

Abstract

Background: Coronary subclavian steal syndrome (CSSS) is an uncommon cause of myocardial ischemia following coronary artery bypass grafting (CABG) utilizing a left internal mammary artery (LIMA) graft. It is most frequently caused by atherosclerotic subclavian artery stenosis, whereas CSSS resulting from thoracic outlet syndrome and dynamic extrinsic compression is exceedingly rare.

Case Presentation: A 66-year-old woman with prior CABG with LIMA to Left Anterior Descending (LAD) and saphenous vein grafts presented 8 years later with recurrent exertional angina despite optimal medical therapy. Coronary angiography revealed proximal subclavian compression with retrograde LIMA flow. Provocative neck rotation during angiography markedly worsened LIMA flow, confirming dynamic extrinsic compression consistent with thoracic outlet syndrome as the underlying mechanism of CSSS.

Management and Outcome: She underwent carotid-subclavian bypass with complete resolution of anginal symptoms. At five-year follow-up, she remained asymptomatic, and nuclear stress testing demonstrated no evidence of inducible ischemia.

Conclusion: This case highlights the importance of provocative positional maneuvers during angiography when evaluating suspected CSSS, particularly when dynamic extrinsic compression is suspected. Identifying the underlying mechanism is critical for appropriate management, and surgical bypass can provide durable long-term symptom relief and restoration of myocardial perfusion in patients with thoracic outlet syndrome-related CSSS.

Keywords: Coronary subclavian steal syndrome; Coronary artery bypass grafting; Thoracic outlet syndrome; Left internal mammary artery

Introduction

Coronary subclavian steal syndrome (CSSS) is a rare but clinically significant complication in patients who have undergone coronary artery bypass grafting (CABG) using a left internal mammary artery (LIMA). Because the LIMA originates from the left subclavian artery, proximal subclavian artery obstruction can compromise coronary blood flow.

CSSS occurs when significant stenosis or occlusion develops proximal to the LIMA origin, resulting in retrograde flow through the LIMA toward the upper extremity and reduced myocardial perfusion [1-2]. Symptoms are typically exertional, as increased upper-extremity demand augments flow reversal and further impairs coronary perfusion, precipitating myocardial ischemia [3].

Atherosclerosis is the most common cause of subclavian artery disease associated with CSSS; however several nonatherosclerotic etiologies have been reported, including Takayasu arteritis, giant cell arteritis, fibromuscular dysplasia, and radiation-induced arteriopathy [1,3-4]. An exceptionally rare cause is thoracic outlet syndrome (TOS), a group of disorders resulting from neurovascular compression at the thoracic outlet. Neurogenic TOS accounts for approximately 95% of cases, whereas arterial TOS represents only 1–5% of cases and is typically caused by extrinsic vascular compression from congenital abnormalities such as cervical ribs or fibrous bands [5]. In arterial TOS, positional maneuvers such as neck rotation or upper-extremity movement may dynamically worsen subclavian artery compression, intensify flow reversal through the LIMA graft, and provoke myocardial ischemia [6-7]. Consequently, diagnosis may be challenging because vascular compromise can be underestimated in the neutral position and become apparent only during provocative testing.

We report a rare case of CSSS secondary to arterial thoracic outlet syndrome with dynamic positional exacerbation demonstrated angiographically. This case highlights the importance of provocative maneuvers during diagnostic evaluation, the need to identify the underlying mechanism of subclavian obstruction, and the durability of surgical revascularization in this uncommon but clinically important condition.

Case Presentation

A 66-year-old Caucasian woman with a history of hypertension, hyperlipidemia, irritable bowel syndrome and multivessel coronary artery disease presented with recurrent exertional chest pain. Eight years earlier, she had undergone three-vessel coronary artery bypass grafting (CABG) with a left internal mammary artery (LIMA) graft to the left anterior descending artery and saphenous vein grafts (SVGs) to the first and second obtuse marginal arteries. During subsequent years, she required multiple percutaneous coronary interventions, including stenting of the left main coronary artery, both vein grafts, and the right coronary artery for progressive atherosclerotic disease and restenosis. Despite these interventions and optimization of antianginal therapy, she continued to experience typical exertional chest pain. She was initiated on medical therapy with ranolazine, isosorbide mononitrate. Six weeks later, her chest pain persisted despite optimal antianginal medical therapy, prompting coronary angiography.

Because of persistent symptoms, repeat coronary angiography was performed. The previously treated coronary vessels and SVGs were patent without significant obstructive disease to explain her symptoms. Selective angiography of the left subclavian artery demonstrated dynamic narrowing proximal to the origin of the LIMA, suggestive of extrinsic compression. During provocative testing, leftward neck rotation resulted in marked worsening of subclavian narrowing with a corresponding reduction in LIMA flow, confirming a positional mechanism consistent with arterial thoracic outlet syndrome causing coronary subclavian steal syndrome (Figure 1A-B). Left coronary angiogram revealed filling of the LIMA retrogradely via LAD noted (Figure 2). The exact anatomic source of compression, such as a cervical rib or fibrous band, was not further characterized by cross-sectional imaging.

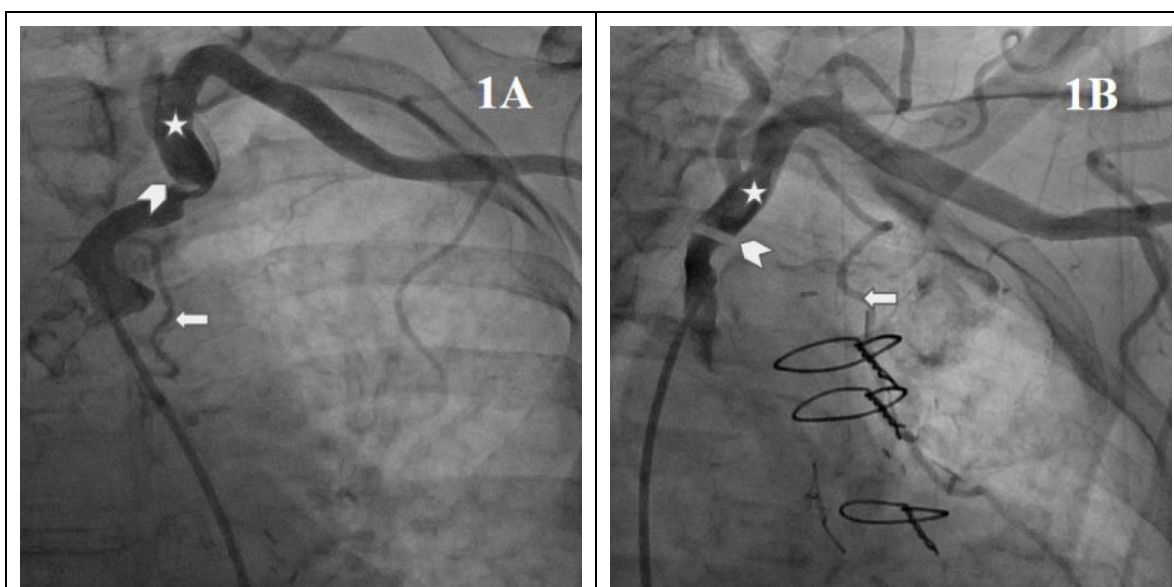


Figure 1 shows the subclavian stenotic lesion from an LAO projection (1A) while figure 1B shows the subclavian artery (star) from an RAO projection showing what appears to be a fibrous or muscular band (arrow head). The LIMA-left internal mammary artery (arrow) fills in both projections with delayed filling.

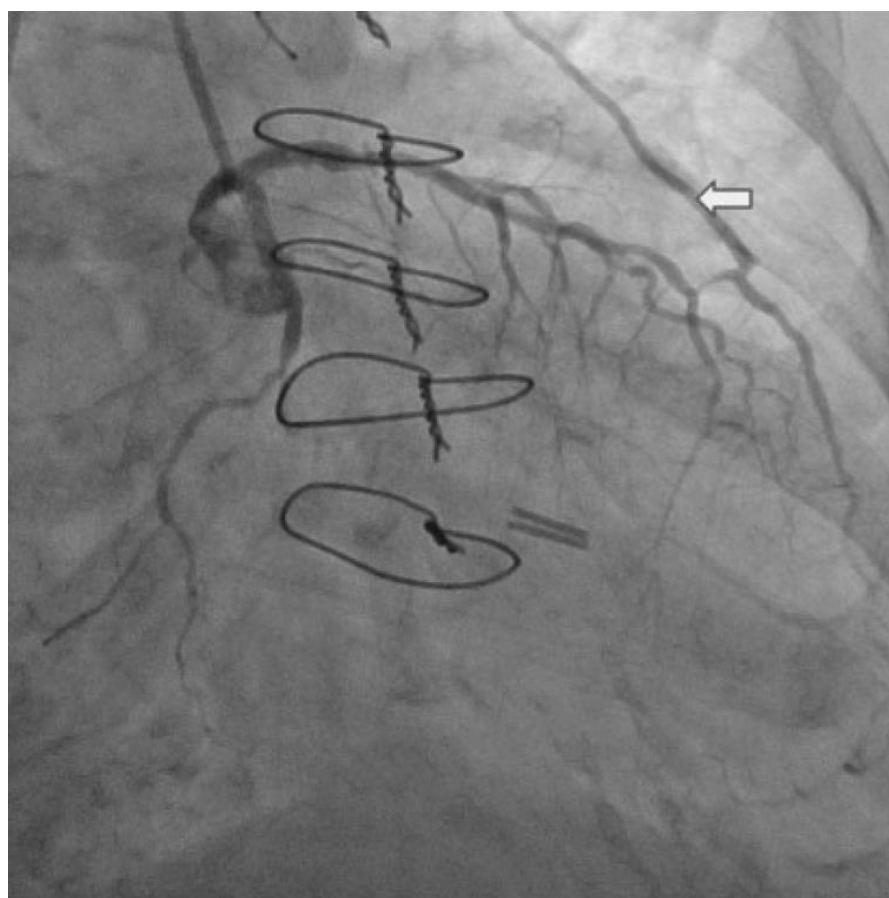


Figure 2 shows the native LAD filling the grafted portion of the distal LAD filling in antegrade fashion and then competitively fills the LIMA graft retrograde (arrow).

Given the dynamic extrinsic nature of the lesion, the patient was deemed a poor candidate for endovascular intervention because angioplasty and stenting would not adequately address the underlying mechanical compression and could predispose to recurrent stenosis or stent-related complications. She was therefore referred for surgical revascularization and subsequently underwent carotid-subclavian bypass using a 6-cm polytetrafluoroethylene (PTFE) graft anastomosed from the left common carotid artery to the subclavian artery distal to the stenotic segment.

Following surgery, her exertional angina resolved completely. At five-year follow-up, nuclear stress testing demonstrated no evidence of inducible myocardial ischemia, supporting long-term graft patency and durable elimination of the coronary steal phenomenon. Although she reported intermittent atypical chest discomfort, no objective evidence of recurrent myocardial ischemia was identified.

Discussion

Coronary subclavian steal syndrome is an uncommon but clinically important complication following CABG with LIMA grafting, with an estimated incidence of approximately 2.5-4.5% [2,8]. Most cases result from proximal subclavian artery atherosclerotic stenosis, which produces retrograde flow through the LIMA graft and compromises myocardial perfusion. It occurs about twice as frequently in men as in women, largely reflecting the predominance of atherosclerotic etiologies [1].

The present case is particularly noteworthy because the mechanism of CSSS was not intrinsic atherosclerotic disease, but rather dynamic extrinsic compression secondary to arterial thoracic outlet syndrome (TOS), an exceedingly rare etiology with limited prior documentation in the literature. TOS encompasses a spectrum of disorders caused by compression of the neurovascular bundle at the thoracic outlet. Although neurogenic TOS accounts for approximately 95% of cases, arterial TOS represents only 1–5% and is typically associated with congenital abnormalities such as cervical ribs or fibrous bands causing subclavian artery compression [5-6]. Given the estimated overall incidence of TOS of only 1–3 per 100,000 individuals, the occurrence of TOS-induced arterial compression leading to CSSS represents an exceptionally uncommon clinical entity.

Diagnosis of CSSS may be particularly challenging because the steal phenomenon is often dynamic and exacerbated by exertion or positional changes, whereas most diagnostic imaging is obtained under resting conditions [1-2,8]. This discrepancy between symptom provocation and diagnostic evaluation may contribute to delayed recognition or underdiagnosis. Our case highlights the critical importance of provocative maneuvers during angiographic assessment. During leftward neck rotation, flow through the LIMA graft visibly worsened, confirming the positional and dynamic nature of the subclavian compression. These findings are consistent with current recommendations for evaluation of arterial thoracic outlet syndrome, which emphasize provocative positioning during vascular imaging to demonstrate dynamic compression and associated hemodynamic compromise [9-11]. Reproduction of physiologic flow limitation during positional maneuvers not only strengthens diagnostic certainty but also provides important mechanistic information that directly influences therapeutic decision-making.

Screening for subclavian artery stenosis in patients with prior CABG remains clinically important, particularly in individuals presenting with recurrent angina despite otherwise adequate coronary revascularization. Bilateral upper-extremity blood pressure measurement remains the simplest and most readily available screening tool [1,3,8]. Significant inter-arm blood pressure differences or diminished upper-extremity pulses should prompt further vascular evaluation. Routine bilateral blood

pressure assessment before CABG may also help identify occult subclavian stenosis and reduce the risk of postoperative coronary steal syndrome. Duplex ultrasonography may demonstrate retrograde vertebral artery flow and, when combined with provocative maneuvers, can help identify dynamic vascular compression [3,10-11]. Additional noninvasive modalities, including computed tomography angiography and magnetic resonance angiography, may further characterize vascular anatomy and extrinsic compression. However, invasive angiography with provocative positioning remains the diagnostic gold standard in suspected TOS-related CSSS because it allows real-time assessment of dynamic flow limitation while simultaneously determining suitability for intervention [9-10]. Although cross-sectional imaging was not performed in our patient to identify the precise compressive structure, the reproducible positional worsening of LIMA flow during angiography strongly supported thoracic outlet-related vascular compression as the underlying mechanism.

Management of CSSS depends primarily on the underlying etiology and anatomic suitability for intervention. Endovascular angioplasty and stenting are generally considered first-line therapy for fixed atherosclerotic subclavian stenosis [3,8,12]. In contrast, our patient required surgical revascularization because the lesion resulted from extrinsic mechanical compression rather than intrinsic vascular disease. In such cases, endovascular stenting alone may be ineffective and potentially susceptible to recurrent compression, restenosis, stent deformation, or fracture [5]. For this reason, carotid-subclavian bypass was favored over percutaneous intervention. Surgical bypass using a polytetrafluoroethylene (PTFE) graft successfully bypassed the compressed arterial segment and restored antegrade flow through the LIMA graft.

Surgical revascularization for subclavian artery disease has demonstrated excellent long-term durability. Reported primary patency rates for carotid-subclavian bypass range from 96–100% at five years, compared with approximately 70–82% for endovascular approaches [3]. Our patient demonstrated sustained symptomatic benefit following surgical intervention, with complete resolution of exertional angina after bypass surgery. At five-year follow-up, repeat nuclear stress testing demonstrated no evidence of inducible myocardial ischemia, supporting continued graft patency and durable resolution of the steal phenomenon. These findings further validate the role of surgical revascularization in patients with mechanically mediated subclavian compression unsuitable for endovascular treatment.

This case highlights several important clinical principles. First, CSSS may occur secondary to rare nonatherosclerotic etiologies, including dynamic extrinsic compression from thoracic outlet syndrome. Second, provocative maneuvers during angiography are essential for identifying positional vascular compression and distinguishing dynamic lesions from fixed atherosclerotic disease. Third, treatment strategies should be tailored to the underlying pathophysiologic mechanism, with surgical bypass favored in cases of extrinsic mechanical compression. Finally, the durable long-term outcome observed in this patient underscores the effectiveness of surgical revascularization for appropriately selected patients with TOS-related CSSS.

Conclusion

This case describes an exceptionally rare presentation of coronary subclavian steal syndrome caused by dynamic subclavian artery compression from arterial thoracic outlet syndrome. Provocative angiographic testing demonstrated positional worsening of flow through the LIMA graft during neck rotation, establishing the diagnosis and identifying the mechanism of recurrent myocardial ischemia.

The case highlights the importance of considering dynamic vascular compression in post-CABG patients with recurrent angina, particularly when conventional coronary evaluation is unrevealing. It also underscores the value of bilateral upper-extremity blood pressure assessment and provocative vascular imaging in detecting occult subclavian pathology. In contrast to atherosclerotic CSSS, which is often managed endovascularly, mechanically mediated lesions may require surgical revascularization. The complete resolution of symptoms and absence of inducible ischemia at five-year follow-up support carotid-subclavian bypass as an effective and durable treatment strategy in appropriately selected patients.

Key Learning Points

- CSSS should be considered in post-CABG patients with recurrent angina and LIMA grafts
- Thoracic outlet syndrome is a rare but important nonatherosclerotic cause
- Provocative positioning during angiography may reveal dynamic compression
- Treatment should target the underlying mechanism
- Surgical bypass provides durable results for extrinsic compression lesions

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