

Case in Point

The Importance of Subclavian Angiography in the Evaluation of Chest Pain: Coronary-Subclavian Steal Syndrome

Menhel Kinno, MD; Osama Tariq Niazi, DO; Jeffrey D. Lorin, MD; and Kulandaivelu Chandrasekaran, MD

Routine preoperative screening for the presence of brachiocephalic disease using ultrasonic duplex or angiography is a cost-effective and essential means to prevent the development of rare occurrences of coronary-subclavian steal syndrome.

Coronary-subclavian steal syndrome (CSSS) is a rare clinical entity with an incidence of 0.2% to 0.7%.¹ Despite its scarcity, CSSS is a condition that can result in devastating clinical consequences, such as myocardial ischemia, ranging from angina to myocardial infarction (MI) and ischemic cardiomyopathy.²

In 1974, Harjola and Valle first reported the angiographic and physiologic descriptions of CSSS in an asymptomatic patient who was found to have flow reversal in the left internal mammary artery (LIMA) graft in a follow-up coronary angiography performed 11 months after coronary artery bypass grafting (CABG).³ Because of the similarity in the patho-

physiology of this condition with vertebral-subclavian steal syndrome, this clinical entity was named coronary-subclavian steal syndrome (CSSS).^{4,5}

In steal-syndrome phenomena, there is a significant stenosis in the subclavian artery proximal to the origin of an arterial branch, either LIMA or vertebral artery, resulting in lower pressure in the distal subclavian artery. As a result, the negative pressure gradient might be sufficient to cause retrograde flow; consequently causing arterial branch “flow reversal,” and then “steal” flow from the organ—either heart or brain—supplied by that artery.^{3,6}

Coronary-subclavian steal syndrome is caused by a reversal of flow in a previously constructed internal mammary artery (IMA)-coronary conduit graft. It typically results from hemodynamically significant subclavian artery stenosis proximal to the ipsilateral IMA. The reversal of flow will “steal” the blood from the coronary territory supplied by the IMA

conduit.^{4,5} The absence of proximal subclavian artery stenosis does not preclude the presence of this syndrome; reversal in the IMA conduit can occur in association with upper extremity hemodialysis fistulae or anomalous connection of the left subclavian artery to the pulmonary artery in d-transposition of the great arteries.² Although the stenosis is most commonly caused by atherosclerotic disease, other clinical entities, including Takayasu vasculitis, radiation, and giant cell arteritis, have been described.⁶ Patients with CSSS usually present with stable or unstable angina as well as arm claudication and various neurologic symptoms.⁵ The consequence of CSSS can include ischemic cardiomyopathy, acute MI,⁷ stroke, and death.^{5,8}

CASE PRESENTATION

A 66-year-old man with a previous MI managed with CABG, permanent atrial fibrillation (AF), and moderate aortic stenosis presented to the ambulatory clinic with

Dr. Lorin is a physician at the Manhattan Campus of the VA NY Harbor Healthcare System and an assistant professor at New York University School of Medicine, both in New York. **Dr. Chandrasekaran** is a physician at the East Orange Campus of the VA New Jersey Health Care System in East Orange, New Jersey. **Dr. Kinno** and **Dr. Niazi** are third-year cardiology fellows at Rutgers New Jersey Medical School in Newark, New Jersey.

recurrent symptoms of stable angina despite being on maximal anti-anginal therapy. A coronary angiogram performed 4 years earlier had revealed significant left main artery disease and total occlusion of the right coronary artery. As a result, CABG was performed with a LIMA conduit to the left anterior descending artery (LAD) and a saphenous venous graft to the posterior descending artery. On physical examination, the patient was comfortable with a heart rate of 70 bpm, blood pressure of 120/80 mm Hg (measured on right arm and no significant difference in blood pressure was reported on the left side).

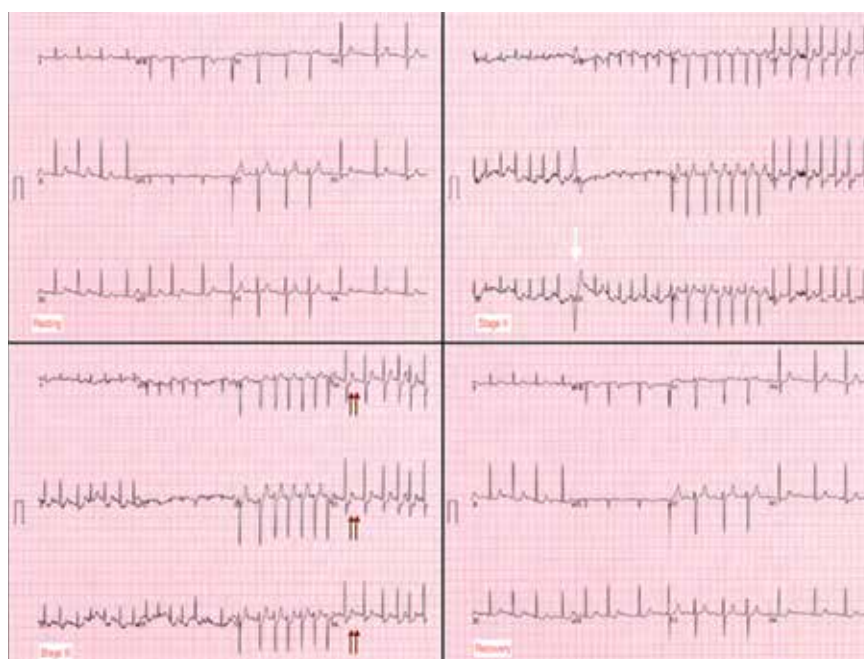
Cardiovascular examination revealed an irregular rhythm with a normal S1, variable S2, and a 3/6 systolic ejection murmur heard best at the right second intercostal space with radiation to the carotids. His peripheral pulses were equal and symmetric in the lower extremities, and no peripheral edema was noted. The remainder of the physical examination was otherwise unremarkable. The resting 12-lead electrocardiogram showed AF at a rate of 60 bpm (Figure 1).

A stress test was performed to elucidate a possible coronary distribution for the cause of the chest pain. The patient was able to exercise for 5 minutes 20 seconds on a standard Bruce protocol, reaching a heart rate of 169 bpm (109% of maximum predicted heart rate) and achieving 7.1 metabolic equivalents. The test was stopped because of dyspnea. The electrocardiogram showed isolated premature ventricular complexes (stage 2, white arrow) and 2- to 3-mm horizontal ST depression at peak exercise (stage III, double arrows) in V4 to V6 that quickly became upsloping and then resolved at rest (Figure 2). Otherwise, the patient had

Figure 1. Resting 12-Lead Electrocardiogram



Figure 2. Standard Bruce Protocol Exercise Electrocardiogram Stress Test



an uneventful recovery period.

Consequently, coronary angiography was performed and showed 95% left main stenosis and total occlusion of the mid-right coronary artery with right dominance, patent LIMA to mid-LAD and patent saphenous

venous graft to posterior descending artery grafts (Figure 3) (Supplemental videos 1, 2, 3, and 4, available at www.fedprac.com). Peripheral angiography showed severe (80%-90%) left subclavian stenosis proximal to the takeoff of the LIMA graft with

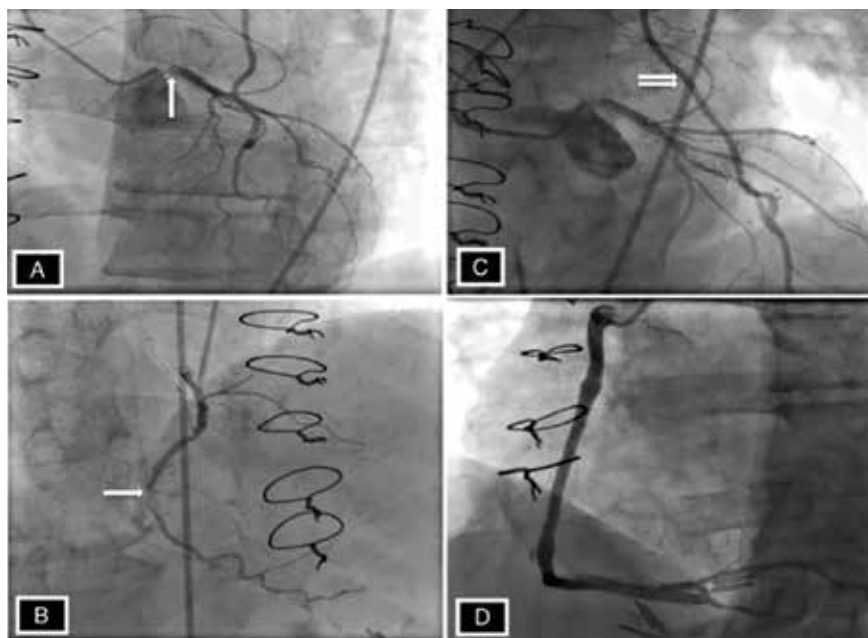


Figure 3. A, Coronary angiography revealed 95% left main stenosis, vertical arrow. B, Total occlusion of the mid-right coronary artery, horizontal arrow, with right dominance. C, Patent left internal mammary artery, double arrows, to mid-left anterior descending artery. D, Patent saphenous venous graft to posterior descending artery grafts.

retrograde flow into the entire LIMA during contrast injection (Figure 4) (Supplemental video 5, available at www.fedprac.com). Given these findings, the patient was diagnosed with CSSS and was referred for intervention.

The patient underwent percutaneous transluminal angioplasty (PTA) of the subclavian stenosis with insertion of an 8 mm x 27 mm balloon-expandable peripheral stent (Figure 5) (Supplemental video 6, available at www.fedprac.com). The patient tolerated the procedure well without complications and with resolution of his symptoms at a 6-month follow-up.

DISCUSSION

Long-term follow-up of LIMA as a conduit to LAD has shown a 10-year patency of 95% compared with 76% for saphenous vein and an associated 10-year survival of 93.4% for LIMA

compared with 88% for saphenous vein graft.^{9,10} Because of the superiority of LIMA outcomes, it has become the preferred graft in CABG. However, this approach is associated with 0.1% to 0.2% risk of ischemia related to flow reversal in the LIMA because of CSSS.²

Greater awareness and improvement in diagnostic imaging have contributed to the increased incidence of CSSS and its consequences.² Although symptoms related to myocardial ischemia, as in this case, are the most dominant in CSSS, other brachiocephalic symptoms, including vertebral-subclavian steal, transient ischemic attacks, and strokes, have been reported.¹¹ Additionally, the same disease might compromise distal flow, resulting in extremity claudication or even distal microembolization.¹²

It is important to recognize that significant brachiocephalic stenosis

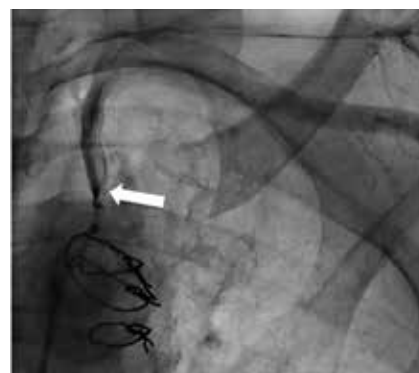


Figure 4. Peripheral angiography showed severe (80%-90%) left subclavian stenosis (arrow).

has been reported in about 0.2% to 2.5% of patients undergoing elective CABG.^{6,8} Therefore, it is essential to screen for brachiocephalic artery disease before undergoing CABG. Different strategies have been suggested, including assessing pressure gradient between the upper extremities as the initial step; CSSS should be considered when the pressure gradient is > 20 mm Hg.

Other strategies include ultrasonic duplex scanning with provocation test using arm exercise or reactive hyperemia.¹³ Many high-volume centers are performing screening by proximal subclavian angiography in all patients undergoing coronary angiography. When significant disease is detected, arch aortography and 4-vessel cerebral angiography is performed.⁶ In addition, other centers have adopted the routine use of computerized tomographic angiography before CABG.¹⁴

Surgical correction of CSSS is considered to be the gold standard and can be accomplished by performing aorta-subclavian bypass, carotid-subclavian bypass, axillo-axillary bypass, or relocation of the IMA graft.² Although this approach is invasive and carries many disadvantages related to patient comfort,

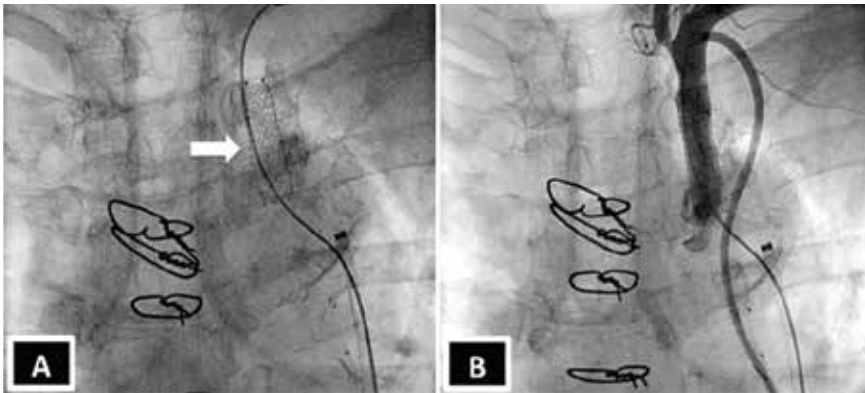


Figure 5. A, Percutaneous transluminal angioplasty of the subclavian stenosis with insertion of an 8-mm x 27-mm balloon-expandable peripheral stent (arrow). B, Subclavian angiography showing improvement in stenosis from 80% to 90% to 0%.

surgical revascularization can be performed safely at the time of CABG and may not carry additional risk of morbidity or mortality.¹⁵ Moreover, surgical correction is the preferred modality for treatment of CSSS when the anatomy is not favorable for percutaneous intervention, such as chronic total occlusion of the subclavian artery.¹⁵ Alternatively, CSSS can effectively be managed less invasively by percutaneous intervention, including PTA with stent placement,^{16,17} thrombectomy¹⁸ or atherectomy of the stenotic subclavian artery.¹⁹

In this patient, PTA was performed with primary stent placement. The lesion was crossed with a sheath, using combined femoral and radial access. After proper positioning, a balloon-expandable stent was deployed that resulted in complete angiographic resolution of the lesion and improvement of symptoms at 6-month follow-up. In line with previous reports, this case demonstrated that percutaneous intervention is a feasible and less invasive approach for management of CSSS.^{16,17} The effectiveness of the percutaneous approach has effectiveness equivalent to surgical bypass with minimal compli-

cations and good long-term success. Therefore, it has been suggested as first-line therapy in CSSS.^{8,16}

Although preoperative screening for brachiocephalic disease before undergoing ipsilateral IMA coronary artery bypass can prevent the development of CSSS, there is controversy about the best approach for managing these concomitant conditions. Many institutions use all-vein coronary conduits, but that forgoes the benefit of a LIMA graft. Therefore, others still perform an IMA conduit after brachiocephalic reconstruction. An alternative method is to use free IMA or radial artery conduit. Currently, there are limited data about the use of endovascular treatment for brachiocephalic disease with a CABG.²

CONCLUSION

Coronary-subclavian steal syndrome is an important clinical condition that is associated with significant morbidity and mortality. In the Sullivan and colleagues report of 27 patients with CSSS, 59.3% had stable angina and 40.7% had acute coronary syndrome, among which 14.8% presented with acute MI.⁷ Therefore, early recognition is essential to pre-

vent catastrophic consequences.

Patients with CSSS usually present with cardiac symptoms, but symptoms related to vertebral-subclavian steal and posterior cerebral insufficiency can coexist. The authors suggest routine preoperative screening for the presence of brachiocephalic disease, using ultrasonic duplex or angiography. This practice is cost-effective and essential to prevent the development of CSSS. Optimal management of brachiocephalic disease prior to CABG is debatable; however, IMA grafting and reconstruction of the brachiocephalic system seems to be a promising approach.

When CSSS develops after CABG, the condition can be successfully treated with percutaneous intervention and outcomes comparable with those of surgical bypass. ●

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