CASE REPORT

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Endovascular treatment of coronary subclavian steal syndrome complicated with STEMI and VF: A case report and review of the literature

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Key Clinical Message

Endovascular treatment of coronary subclavian steal syndrome is usually successful and safe. However, it might be lethally complicated, such as our case of STEMI and ventricular fibrillation during stent deployment. Early diagnosis of subclavian stenosis, intermittent dilations of balloons during stent deployment, and choosing the accurate stent size are suggested to avoid such complications.

KEYWORDS

complication, coronary subclavian steal syndrome, endovascular intervention, resuscitation, STEMI, ventricular fibrillation

1 INTRODUCTION/BACKGROUND

Stenosis of the subclavian artery (SA) can be silent, or it can be the cause of various clinical presentations, such as neurological symptoms of Subclavian Steal Syndrome, or the rare, yet increasingly prevalent, coronary subclavian steal syndrome (CSSS). Currently, its prevalence among coronary artery bypass graft (CABG) patients is approximated to 0.2% to 6.8%.¹

Coronary artery bypass graft with left internal mammary artery (LIMA) graft can be a vascular risk factor that may expose patients to SA stenosis, resulting in stealing from both the vertebral artery (VA) and the LIMA, and subsequently stealing from the coronary circulation, thus decreasing the needed blood supply to the heart muscle, resulting in a variety of sequelae, ranging from no symptoms, to unstable angina, and in as some reported cases, myocardial infarction (MI).²⁻⁴

The most common treatment of CSSS is endovascular intervention and stenting of the stenotic subclavian, it is considered to be a successful procedure that rarely yields significant complications. However, here we describe a case with Intervention on a stenotic SA in a CSSS patient that was complicated by ST-elevation myocardial infarction (STEMI) and ventricular fibrillation (VF). Our case is the first in the literature to describe such events.

CASE PRESENTATION 2

We describe a 74-year-old man with a known history of coronary artery disease and a triple bypass {Saphenous vein graft (SVG)-right coronary artery (RCA), SVG-circumflex (Cx)/obtuse marginal (OM), Left internal mammary artery (LIMA)-left anterior descending artery (LAD)} in 1990. His past medical history is also significant for ischemic cardiomyopathy with ejection fraction of 40%-45%, mild mitral regurgitation (MR), obstructive sleep apnea (OSA), hypertension (HTN), hyperlipidemia (HLD), chronic kidney disease (CKD), and peripheral arterial disease (PAD).

His last percutaneous coronary intervention (PCI) was done in 2012 for a non-ST-elevation myocardial infarction (NSTEMI) that occurred one day after lumbar laminectomy. The PCI showed him to have no native circulation, all three major native coronary arteries were 100% occluded, there was a patent saphenous vein graft to the obtuse marginal, the LIMA graft to LAD was patent with no significant disease

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FIGURE 1 Anterolateral ST-T changes suggestive of ischemia

and good collaterals to circumflex and RCA distal branches. The graft to the RCA was totally occluded.

During 2017, the patient was suffering from crescendo angina and dyspnea. In February of 2018, a nuclear stress study was done and was found to be significantly abnormal. Specifically, the stress ECG exacerbated the patient's symptoms and showed an anterolateral ST-T changes suggestive of ischemia (Figure 1), while the pictures of nuclear study showed multiple defects (inferior and infero-basal partially reversible defects and a distal septal defect which is likely a scar) but was not specific to the pathology that was discovered later. In the light of these findings, a decision was made to perform cardiac catheterization and coronary angiography.

2.1 | Procedure

The procedure started with no problems in access, and the diagnostic catheter (5 Fr JL4) engaged the left main coronary artery with no difficulties, images were taken and showed distal occlusion of the left main artery, an occluded anterior descending artery, and occluded Circumflex/OM branches, SVG-Cx OM and LIMA-LAD were patent. 5 Fr 3DRC catheter was used to engage the native RCA, SVG to OM, then SVG to RCA, images taken showed a proximally occluded right coronary artery, SVG-RCA was occluded as well.

Next, a 3DRC was used to engage the left subclavian artery. Severe occlusive calcific stenosis (95%) was noted (Figure 2). A Glidewire was used to cross the stenosis and the 3DRC was advanced over it. Pressure gradient of 45 mm Hg across the lesion was noted.

After reviewing the images, subclavian intervention was deemed necessary. In view of severe peripheral arterial disease (PAD), no mechanical support was feasible.

With the Glidewire advantage in place, the 5 Fr Sheath was exchanged for a 7 Fr 90 cm Shuttle Sheath which was positioned in the descending thoracic aorta. A powerFlex 4 mm



FIGURE 2 Severe occlusive calcific stenosis (95%) was noted in the left subclavian artery

 \times 20 mm balloon was advanced to the lesion and inflated to 10 ATM x 3 for 20-30 seconds to predilate the lesion.

Next, a LifeStream 8×37 mm covered stent was positioned across the lesion. The stent was deployed for 40 seconds at 13-14 atmospheric pressure to secure full deployment and apposition to vessel wall (Figure 3), after which the patient became hypotensive, anterior ST elevation and VF ensued requiring direct current cardioversion DCCV x 2. There was a brief cardiopulmonary resuscitation (CPR) performed (30 seconds), epinephrine and IV amiodarone were administered. (Follow ECG Tracing in Figure 4).

Selective angiography of the subclavian demonstrated a minor distal dissection (class I). Another LifeStream 8×26 mm stent was deployed distally, sparing the vertebral artery ostium and overlapping the previous. The stents were



FIGURE 3 Endovascular intervention on the stenotic portion of the subclavian artery

post dilated with a PowerFlex 9×20 mm balloon. Final angiography (Figure 5) demonstrated excellent stent expansion and patent branch vessels.

2.2 | Postoperative course

The patient remained somewhat hypotensive, left ventricular end diastolic pressure (LVEDP) was 7 mm Hg. IV fluid bolus of normal saline (2 L) was given, epinephrine drip started. Hemoglobin repeat was 11.9.

A bedside echo demonstrated collapsible inferior vena cava (IVC) indicative of hypovolemia. Epinephrine drip was continued, and blood pressure was maintained in normal range for the next few hours. Patient was chest pain free.

After two days the patient was discharged from the hospital on dual antiplatelet medications.

Two weeks later, follow-up at the clinic patient reported improved shortness of breath and denied angina pectoris.

3 | **DISCUSSION**

Subclavian artery (SA) as other arteries can be subjected to atherosclerosis; in fact, 3%-4% of general population can be affected by subclavian artery stenosis (SAS).⁵ SAS patients can be asymptomatic, but there is portion of them who

develop the well understood steal phenomenon. They can be asymptomatic or may develop a spectrum of manifestations of the subclavian steal syndrome (SSS) ranging from upper extremity claudication and muscular fatigue to vertebrobasilar hypoperfusion resulting in vertigo, diplopia, and syncope.⁵

Coronary subclavian steal syndrome (CSSS) is an uncommon consequence of coronary artery bypass graft with LIMA. Patients with CSSS were first described in 1966 and now incidence rate is 0.2%-6.8%.¹ The incidence is continually rising due to the increased number of CABG patients along with their longer life expectancy. In CSSS, SAS is proximal to the LIMA; therefore, the steal phenomenon can affect both the vertebral artery as well as the LIMA. Exertion of the left upper extremity could divert the blood away from the coronary circulation. Symptoms can include manifestations of SSS and those of myocardial ischemia such as angina pectoris, and rarely, myocardial infarction if the stealing is severe.²⁻⁴ The mean time between CABG surgery and development of symptoms of CSSS is about 9.0 ± 8.4 years.⁶ In our case, the patient had his CABG surgery in 1990 and he started to have symptoms of angina and dyspnea in 2017. He did not manifest the other neurological symptoms of SSS, nor any upper extremity claudication.

Suspicion of SAS should be raised if discordance in blood pressure readings on right and left arms (>15 systolic blood



ECG tracing of the patient in baseline, then to STEMI and VF and resuscitation, then to baseline again FIGURE 4

pressure) and left subclavian bruits and were present.⁷ To confirm the diagnosis of this pathology and explore its consequences, a variety of methods could be utilized, such as Doppler ultrasonography, nuclear stress test, CT angiography (CTA), MRI angiography (MRA), and the conventional catheter angiography. This approach is beneficial in both CSSS and pre-CABG patients.8

Doppler ultrasonography is a well-established noninvasive diagnostic tool with a relatively low cost, which make it favored over CTA and MRA to be used in evaluating blood flow in both LIMA and SA.9 Limitations would be the visualization difficulty due to the overlying bony structures, and the fact that severe stealing phenomena might be necessary for the test to yield significant results. Therefore, it might be beneficial to induce hyperemia in the ipsilateral upper extremity by inflating a blood pressure cuff to suprasystolic pressure for five minutes followed by deflation to uncover steal ultrasonographic findings.⁸

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Nuclear stress test acquires sensitivity of 80%-90% and specificity of 70%-80% in diagnosing coronary artery disease,¹⁰ and it proved its value in diagnosing CSSS in prior documented cases by revealing ischemia of the myocardium in the regions that are perfused by the LAD.^{11,12}

Preprocedural CTA can be used to construct an anatomic map of lesion morphology, location, and length, after which,





FIGURE 5 Final angiography demonstrated excellent stent expansion and patent branch vessels

MRA would be proper to view the flow direction, therefore confirming the diagnosis of CSSS. These modalities facilitate in building a better management plan. In stenotic lesions, they would aid in choosing the appropriate endovascular intervention access, along with the stent choice of type and size. While in occluded lesions, they would direct toward a surgical bypass approach as a safer treatment.^{9,13}

Despite the presence and introduction of these multiple methods of diagnosis, conventional angiography is still considered the gold standard for evaluation of LIMA function and reversal flow detection.⁷ However, using angiography to evaluate SA in pre-CABG patients is still controversial, even with the presence of clinical signs of SAS, and that is due to possible complications that might make its risks outweigh the benefits.8

In our patient, a discrepancy of BP between the two upper extremity was noted, but a bruit was not appreciated during physical examination, most probably due to the severity of the lesion. Therefore, we approached the case by a more traditional accessible diagnostic methods of nuclear stress test and conventional angiography. The stress exam exacerbated the patient's symptoms and managed to reveal an accurate information on the ischemic region being in the anterolateral myocardium (LAD perfusion segments), while the nuclear images reported different nonspecific results. After that, we performed an angiography which showed distal occlusion of the left main artery, an occluded LAD, occluded LCx/OM branches, patent SVG-Cx OM and LIMA-LAD grafts, proximally occluded RCA and SVG-RCA graft. Also, severe occlusive calcific stenosis in the left subclavian (95%) was noted. These findings meant about >90% of

the circulation to the myocardium depended solely on the LIMA-LAD graft.

Coronary subclavian steal syndrome has two methods of treatment, endovascular manipulation and surgery. Although till now, no randomized trials have been performed to compare the efficacy of one strategy versus another,¹⁴ the percutaneous revascularization seems to be at least equivalent to surgery in terms of effectiveness with less procedure-related serious complications.¹⁵ It was stated in the 2011 ACC/AHA guidelines that surgical and endovascular revascularization can both be considered as first-line treatment of SA stenosis. Nevertheless, in 2011, The European Society of Cardiology stated that the first line of management of SA stenosis is percutaneous balloon angioplasty with stent support, and surgical intervention takes place in case of PTA/Stent failure. The general preference of endovascular treatment is related to avoidance of general anesthesia, lower peri-procedural morbidity and mortality, and lower hospital stay. However, the risk of severe complications should not be underestimated. particularly in the case of calcific subclavian lesions.⁶ Those complications include dissections, coronary hypoperfusion, heart failure, and hemodynamic collapse.¹⁶

Here, in this case, after the angiogram, we decided that treatment is necessary and cannot be postponed due to the patient's dependence on blood supply from the SA, and endovascular manipulation was a relatively safe and acceptable choice. The lesion was not totally occluded, the EF was 40%-45%, and there was no need for a mechanical support device. Our intervention access was through the femoral artery, due to its acceptance of a larger sheath size while providing an easier more accessible management technique considering the urgency of the situation. Our stent choice was of a balloon-expandable rather than the self-expandable because of the need to dilate the significantly large calcified stenotic lesion using the balloon, and because of the higher radial strength that can sustain the critically needed dilation of the artery. Upon deployment of the stent, the patient became hypotensive, ST elevations in the anterior leads were noticed and this was followed by immediate VF requiring 2xDCCV and a brief CPR after which the patient recovered. Moreover, angiography following the incidence showed a minor dissection (class I), and this required deployment of another overlapping stent. Apart from the fact that the patient remained hypotensive after the procedure and needed IV fluids for few hours, he recovered fairly well, and his symptoms ceased. During two follow-ups since the procedure, he was found to be completely asymptomatic.

STEMI and VF in this case can be explained by temporary occlusion of SA during deployment of the first stent compromising blood supply through the LIMA-LAD graft on which he was solely dependent with absent native circulation.

It is important to shed some light on the fact that the technical success rate for endovascular treatment of SA stenosis

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Angle et al (2003) ¹⁸ 18 patients with NA Patient dev	Dilatation with cutting SA spiral d balloon (4.0 3 15 mm, and subsec Flextome Cutting LIMA gra Balloon, Natick, Mass) occlusion use of cutt balloon fo predilatati	lissection Prolonged balloon inflation of SA was equent to restore blood flow along with PCI of aft native LAD (incompletely occluded) t after the enhance coronary circulation. LIMA g ting was lost.	 Dlied • Use cutting balloon cautiously in heavily calcified lesions. ft
endovascular angina du intervention, four procedure developed complications consequentially, 1/4 is significant for this study	NA Patient dev angina du procedure	 (eloped Quickly resolved after administration o uring the sublingual nitroglycerin, then 4 d later underwent a successful previously pla CABG (repeat CABG) 	 Routine screening for left SA stenosis in patients with LIMA grafts. Possible need of distal protection against intracranial and coronary embolization. Enhancement of brachial artery flow by provocative maneuvers may be of benefit in preventing intracranial or coronary embolization.

TABLE 1 Literature review of cardiac complications of endovascular treatment of coronary subclavian steal syndrome

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is almost 100%.¹⁷ Moreover, complications are very rare, most notably due to high technical skills and experience in endovascular interventions. Also delayed reversal of LIMA blood flow would protect the coronary arteries from micro embolism.¹⁷ The literature mentions some complications which can be divided to those which may occur in any endovascular manipulations (hematomas), those that can occur in any subclavian manipulation (TIAs) and very rarely those which affect the coronary circulation. Upon a comprehensive review of more than 170 cases in the literature, we found two studies which reported dissections that compromised the LIMA graft, one of which led to total occlusion. The third study reported severe angina pectoris during the intervention. These studies warned against the possibility of complications and suggested some recommendations to avoid such events. We summarized these findings in the attached Table 1. Our case is the first in the literature to report almost a lethal complication of STEMI and VF in endovascular treatment of CSSS.

To prevent such a serious condition, we suggest the use of short intermittent gradual dilation during balloon and stent implantation in patients who depend on LIMA for their coronary circulation. (Ten seconds repeated inflations to nominal pressure until less than rated pressure achieved), this will avoid risky prolonged interruption of blood supply through the SA and LIMA to LAD graft. Moreover, choosing the appropriate size of the stent plays an important role in preventing dissection which can compromise flow to LIMA graft. In our case, oversized stent was probably one of the factors that led to dissection. Also, the interventionist should be fully aware that multiple high-pressure dilations can alter the atherosclerotic plaque and possibly cause an embolization.¹⁷ We can also use cardiac support devices such as Impella¹⁶ in patients with poor LV function, which would limit and prevent myocardial damage and cardiogenic shock. We also emphasize the importance of physical examination and measurement of blood pressure in both upper extremities, a difference greater than 20 of SBP may suggest subclavian stenosis. Lastly, assessing the degree of stenosis by the proper diagnostic methods and application of the appropriate method of treatment can protect from various possible complications.

4 | CONCLUSION

Even though CSSS is rare, cases are increasing in number with time. Therefore, diagnosing and treating SA stenosis is increasingly of greater importance. Although endovascular treatment became popular for its safety and fast recovery; we should be aware that lethal complications such as VF and hemodynamic collapse are possible risks. Early diagnosis of subclavian stenosis, intermittent dilations of balloons during stent deployment, choosing the accurate size of the stent, along with other safety measures should be taken to achieve the best outcomes with minimal risks.

CONFLICT OF INTEREST

None declared.

AUTHOR CONTRIBUTION

MHB: reported the case and reviewed its literature, collected the patient's data, procedure details, and specifics of the complication and its resolution, reviewed literature for findings and information about CSSS, drafted the paper, obtained the appropriate informative figures, and involved in the communication with journal, and re-editing of the paper as per their requests. MN: performed the endovascular intervention in our case, overseen the work process and planned the time lines, provided the necessary information about the specific technicalities of the intervention along with the case's deductions, interpretations, and suggested editing's. LAS: obtained patient's informed consent to write his case, contributed in organizing patient's data and the literature review, assessed in drafting the paper, revised and re-edited as per suggestions.

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