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CASE SERIES
Coronary heart disease

Endovascular treatment of coronary subclavian steal syndrome: a case series highlighting the diagnostic usefulness of a multimodality imaging approach

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Background Coronary subclavian steal syndrome (CSSS) is an uncommon complication observed in patients after coronary artery bypass surgery with left internal mammary artery (LIMA) grafts. It is defined as coronary ischaemia due to reversal flow from the LIMA to the left subclavian artery (SA) when a proximal left SA stenosis is present. In practice, the entire clinical spectrum of ischaemic heart disease, ranging from asymptomatic patients to acute myocardial infarction, may be encountered.

Case summary Three cases of CSSS recently detected at our hospital are being described. Two patients presented with an acute coronary syndrome, so diagnosis was suspected based on coronary angiography findings, as retrograde blood flow from LIMA to the distal SA was present. Myocardial ischaemia was documented by myocardial perfusion scintigraphy in one case. The third patient was asymptomatic and CSSS was suspected during physical examination and confirmed by computed tomography (CT). Endovascular intervention with balloon-expandable stent implantation of the stenotic SA was performed by vascular surgeons in all patients. No periprocedural complications occurred, and complete resolution of symptoms was achieved.

Discussion In CSSS, subclavian angiography is the standard diagnostic test. However, other diagnostic techniques may be valuable to better clarify this challenging diagnosis. In the herein small series, the usefulness of a multimodality imaging approach including Doppler ultrasound, myocardial perfusion scintigraphy, and CT is well demonstrated. Furthermore, this study endorses the safety and utility of endovascular treatment in different clinical scenarios, including asymptomatic patients.

Keywords Coronary artery bypass surgery • Coronary subclavian steal syndrome • Subclavian stenosis • Acute coronary syndrome • Myocardial ischaemia • Case Series

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Learning points

- A high index of clinical suspicion of coronary subclavian steal syndrome (CSSS) in patients with coronary artery bypass graft surgery and severe peripheral artery disease presenting with myocardial ischaemia should be maintained.
- A multimodality imaging approach is recommended to clarify this challenging diagnosis.
- This case series supports the safety and utility of endovascular treatment with balloon-expandable stents in patients with CSSS.

Introduction

Coronary subclavian steal syndrome (CSSS) is a severe and rare complication observed after coronary artery bypass graft surgery (CABG) with left internal mammary artery (LIMA) grafting. It is defined as myocardial ischaemia resulting from the reverse blood flow from LIMA graft to left subclavian artery (SA).[1] This uncommon syndrome represents a diagnostic challenge due to its silent or non-specific clinical presentation. According to the European and American guidelines,[2,3] percutaneous balloon angioplasty with stent implantation is recommended as first-line treatment of SA stenosis. We report three CSSS cases with different clinical presentations, highlighting the importance of a high index of clinical suspicion, advantages of a multimodality imaging diagnostic approach, and safety of endovascular treatment.

Timeline

Patient 1

- February 2016: non-ST-elevation myocardial infarction. Coronary artery bypass graft surgery (CABG): left internal mammary artery (LIMA) to left anterior descending artery (LAD) and sequential right internal mammary artery (RIMA) from LIMA to ramus intermediate branch and obtuse marginal artery.
- July 2019: admission with new-onset angina pectoris.
- Day 4 of admission: anterior coronary ischaemia in myocardial perfusion scintigraphy.
- Day 7 of admission: diagnosis of left subclavian artery (SA) stenosis with invasive coronary angiogram.
- Day 11 of admission: chest computed tomography (CT) angiogram, where significant stenosis in both common carotid arteries as well as severe left SA stenosis was documented.
- Day 13 of admission: balloon-expandable endoprosthesis implantation.
- Day 15 of admission: discharge.
- December 2019: clinical follow-up, the patient remained asymptomatic.

Patient 2

- 2012: stenting to left iliac artery.
- February 2019: CABG with LIMA to LAD and saphenous venous graft from LIMA to obtuse marginal artery.
- November 2019: admission with acute coronary syndrome. Invasive coronary angiography was performed and left SA stenosis was documented.
- Day 3: balloon-expandable endoprosthesis implantation in left SA stenosis.
- Day 11 of admission: discharge.
- January 2020: clinical follow-up, the patient remained asymptomatic.

Patient 3

- January 2016: endarterectomy to left internal carotid artery (70% stenosis).
- May 2018: CABG with LIMA to LAD and RIMA to second obtuse marginal artery.
- January 2019: left SA stenosis was clinically suspected during vascular surgery consulting, Doppler ultrasound documented retrograde flow in the left vertebral artery.
- January 2019: left SA stenosis confirmed by chest CT angiogram.
- February 2019: successful treatment of left SA stenosis with balloon-expandable stent implantation.
- Day 2 of admission: discharge.
- April 2019: optimal interventional result on chest CT angiogram.
- February 2020: clinical follow-up, the patient remained asymptomatic.

Case presentation

Patient 1

A 61-year-old woman presented to the emergency department with new-onset angina pectoris. She had a history of heterozygous familial hypercholesterolaemia, carotid artery stenosis, and CABG 3 years before the current episode (LIMA to left anterior descending artery (LAD), and sequential right internal mammary artery (RIMA) from LIMA to a ramus intermedius branch and left marginal artery). Medications before admission were losartan, amlodipine, bisoprolol, acetyl salicylic acid (ASA), ticagrelor, omeprazole, and atorvastatin. She was admitted to our hospital with unstable angina (recurrent episodes of angina on minimal effort or at rest). Blood pressure was 102/45 mmHg and heart rate was 66 beats per minute. Electrocardiographic extensive ST-segment depression was clearly documented (*Figure 1*), but there were no segmental wall motion abnormalities on echocardiography and troponin values were 0.04 ng/mL (reference values ≤ 0.05 ng/mL). Renal function was normal (creatinine level 1.05 mg/dL; reference values 0.7–1.3 mg/dL). Physical examination showed weak pulses in the left arm and a left supraclavicular bruit. Gated myocardial perfusion scintigraphy with 99mTc-methoxyisobutylisonitrile (99mTc-MIBI gated-SPECT) was performed to demonstrate the presence and extension of

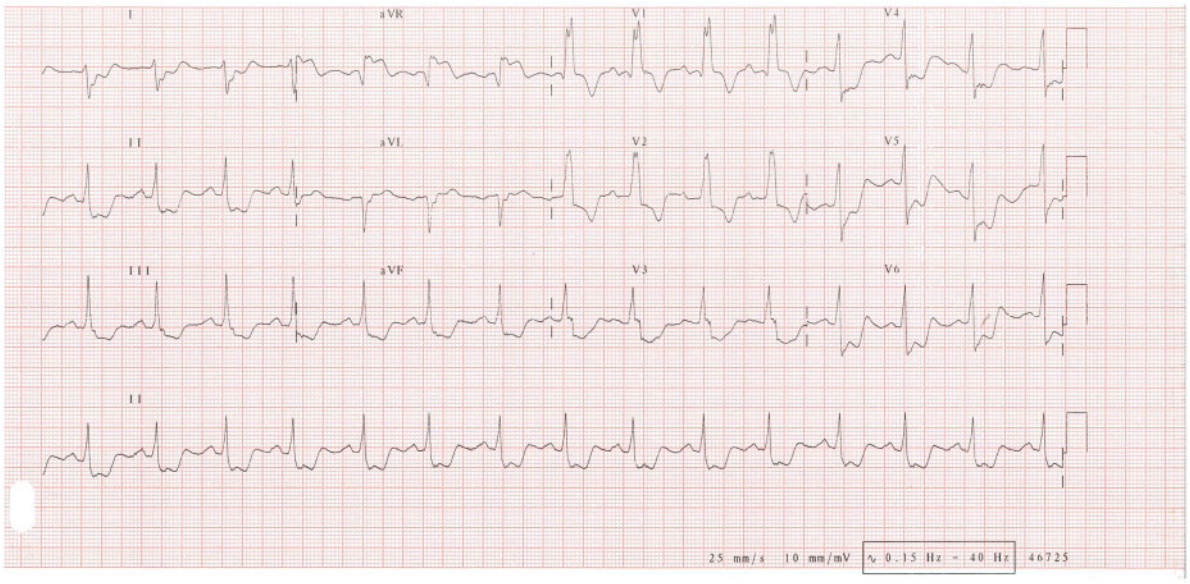


Figure 1 Electrocardiogram of Patient 1. It shows a sinus rhythm and a wide QRS with right bundle brunch block, with right axis deviation. The most important finding in this electrocardiogram is the extensive ST-segment depression in almost all leads, and the ST-segment elevation in leads aVR and V1. These findings are suggestive of diffuse myocardial ischaemia.

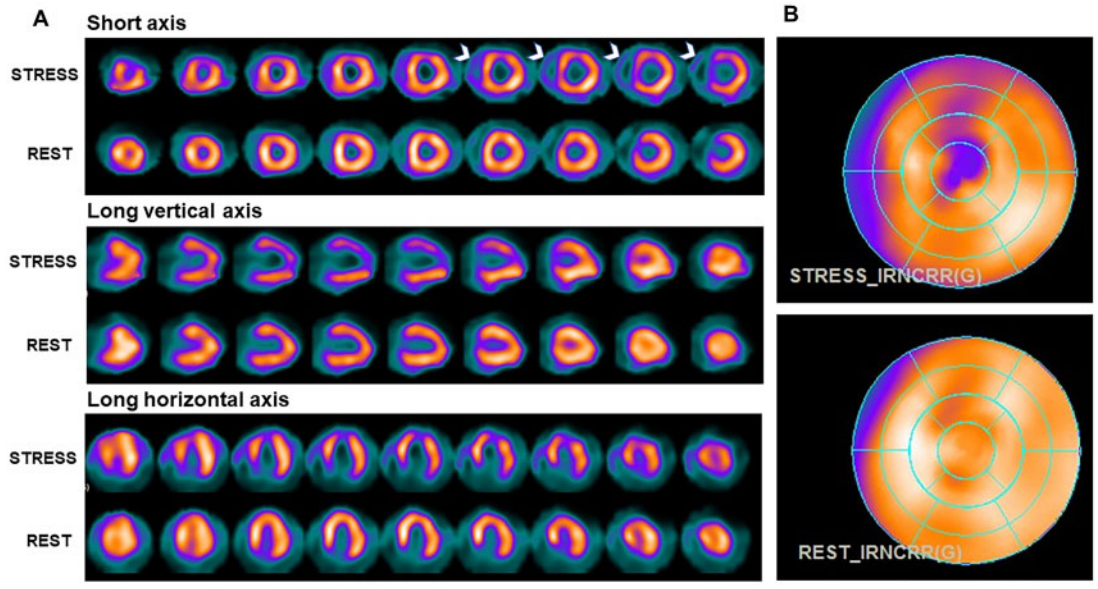


Figure 2 ^{99m}Tc-MIBI gated-SPECT/CT in Patient 1. Tomographic (*A*) and polar maps (*B*) perfusion images obtained during stress and at rest showed a reversible perfusion defect suggestive of mild/moderate ischaemia extending through anterior wall, upper septum, and apex. Post-stress and rest left ventricular ejection fraction were 47% and 70%. Note the most prominent right ventricle cavity after regadenoson stress (arrowheads).

myocardial ischaemia. During vasodilator stress, the patient had chest pain without ST-T changes. Gated-SPECT (non-corrected) images revealed a reversible hypoperfusion in LAD territory and several high-risk functional markers as biventricular dilation and left ventricular systolic dysfunction observed after pharmacological stress (*Figure 2*). Invasive coronary angiography showed no changes in native coronary arteries. A retrograde blood flow was observed within both internal mammary artery grafts (*Figure 3A*; *Video 1*), and contrast

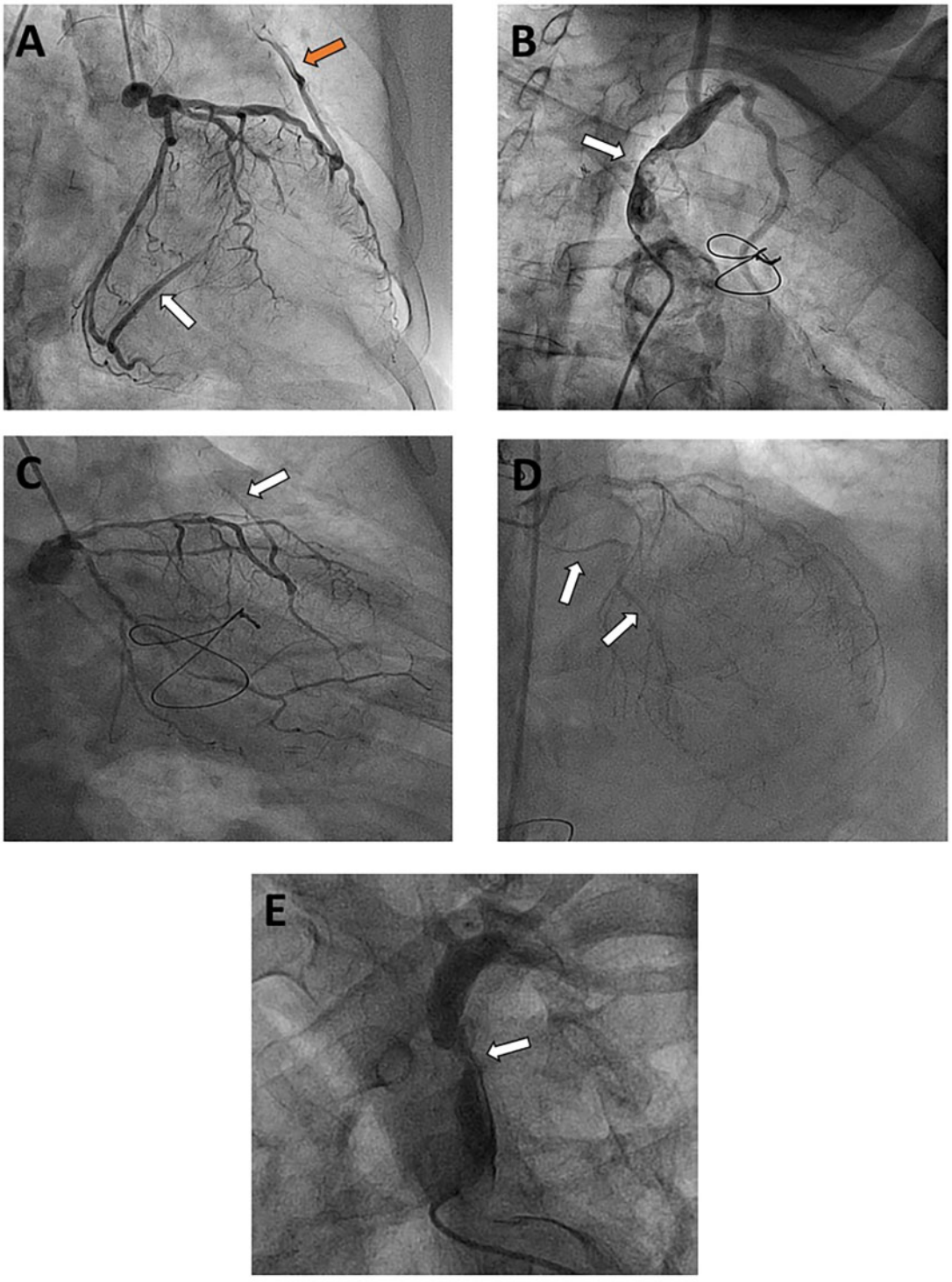
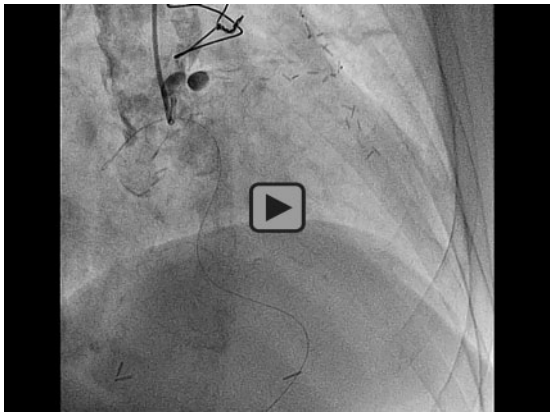
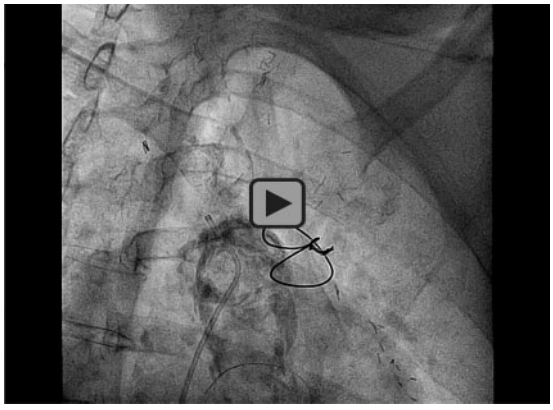


Figure 3 Angiography of Patient 1 (*A* and *B*) and Patient 2 (*C*–*E*). (*A*) This shows retrograde flow within right (white arrow) and left (orange arrow) internal mammary artery grafts. Severe left subclavian artery stenosis is seen in *B* (arrow). (*C* and *D*) Retrograde flow through left internal mammary artery (*C*, arrow) and saphenous venous (*D*, arrows) grafts in Patient 2. (*E*) Severe left subclavian artery stenosis (arrow).

injection into left SA confirmed a proximal severe stenosis (*Figure 3B*, *Video 2*). To better assess the ascending aorta and aortic arch branches, a chest computed tomography (CT) angiogram was performed. An extensively calcified ascending aorta, significant stenosis in both common carotid arteries as well as severe left SA stenosis was documented (*Figure 4A*). In a multidisciplinary heart team meeting, we decided to perform an endovascular SA repair. From right femoral and left axillary approach, a retrograde injection into the SA



Video 1 Coronary angiograma of patient 1. Retrograde flow within both internal mammary artery grafts is observed.



Video 2 Contrast injection into left subclavian artery during coronary angiography shows a proximal severe stenosis in patient 1.

confirmed the stenosis severity (*Figure 5A*). Pre dilatation (Mustang balloons, 5 and 6 mm) was associated with electrocardiographic ST-segment elevation. Then, a balloon-expandable endoprosthesis (Gore Viabahn VBX, 8 × 29 mm) was deployed along the stenotic lesion. Post-procedural angiography (*Figure 5B*) and chest CT angiogram (*Figure 4B*) demonstrated the optimal SA dilatation with patency of LIMA and vertebral artery. There were no periprocedural complications. The patient was discharged 48 h after the procedure. The antithrombotic regimen chosen was dual antiplatelet therapy with ASA and ticagrelor for a duration of 1 year and single antiplatelet treatment with ASA afterwards. In the last medical follow-up, 5 months after hospital discharge, the patient is asymptomatic and physical examination showed symmetrical pulses in both arms and the supraclavicular bruit was not detected.

Patient 2

A 72-year-old man was admitted to the emergency department with a non-ST-elevation myocardial infarction (NSTEMI). He had a history of type 2 diabetes, chronic kidney disease, stent implantation in the left iliac artery 7 years previously, and chronic coronary artery disease treated with drug-eluting stents in the left circumflex artery 14 years ago, and in the right coronary artery 4 years ago. Five months before the current episode, the patient underwent CABG (LIMA to the LAD and a saphenous vein graft from LIMA to the left marginal artery). He was treated with ASA, ticagrelor, omeprazole, atorvastatin, valsartan, bisoprolol, and insulin before the current admission. He presented to the emergency department with prolonged chest pain. Physical examination was normal, and serial electrocardiograms did not show T wave or ST changes. Serum high-sensitivity troponin T level of 3270 ng/L (reference values ≤ 14 ng/L) confirmed the diagnosis of NSTEMI. Creatinine level was 1.9 mg/dL. An invasive coronary angiogram was performed and retrograde flow in both grafts was documented (*Figure 3C* and *D*, *Video 3*, Supplementary material online, *Video S1*). A 50-mmHg pressure gradient was found across the proximal segment of the left SA. Left SA contrast injection confirmed the stenosis severity (*Figure 3E*, Supplementary material online, *Video S2*). An endovascular treatment was decided in a multidisciplinary heart team. In this case, a left brachial artery approach was chosen. A Gore Viabahn VBX balloon-expandable endoprosthesis (8 × 29 mm) was implanted in the stenotic lesion (*Figure 5C*) and post-dilatation was performed with a Mustang balloon (7 mm). The success of the procedure was angiographically confirmed (*Figure 5D*). There were no periprocedural complications and the patient was discharged 8 days after admission taking dual antiplatelet therapy with the intention of withdrawing clopidogrel after 12 months. Six months later, the patient is asymptomatic.

Patient 3

A 76-year-old man was seen in a follow-up visit. He had a history of arterial hypertension, left internal carotid artery stenosis treated with endarterectomy 3 years ago, and CABG 1 year prior to the current consultation (LIMA to the LAD and RIMA to a marginal artery). The patient was asymptomatic. He was taking ASA, atorvastatin, omeprazole, and bisoprolol. Physical examination during follow-up medical consultation revealed the absence of pulses in the left arm and normal pulses in the other limbs. A systolic blood pressure difference was found in the upper extremities (right arm: 170 mmHg, left arm: 132 mmHg; index: 0.78). A Doppler ultrasound was performed to assess the aortic arch branches detecting a retrograde flow in the left vertebral artery. Chest CT angiogram documented an 80% left SA stenosis (*Figure 4C*). Renal function was normal (creatinine level was 0.78 mg/dL). Electrocardiogram showed sinus rhythm without signs of acute ischaemia. Considering the high probability of developing future symptomatic myocardial ischaemia, we decided to perform an endovascular treatment. In this patient, a left brachial and right femoral artery approach was chosen. Retrograde subclavian injection confirmed a severe left SA stenosis (*Figure 5E*). Pre dilatation with a Mustang balloon (6 × 40 mm) was performed, and a balloon-expandable stent (Visipro 8 × 37 mm) was implanted in the SA at the site of the stenosis. Post-procedure angiographic result was optimal (*Figure 5F*) with no complications The blood pressure index between the upper limbs after the procedure was 0.98. The patient was discharged 24 h after the procedure on treatment with ASA and clopidogrel. Clopidogrel was withdrawn 6 weeks later. A new chest CT angiogram performed 2 months after hospital discharge showed absence of stenosis in the left SA (*Figure 4D*). The last follow-up visit at the outpatient clinics

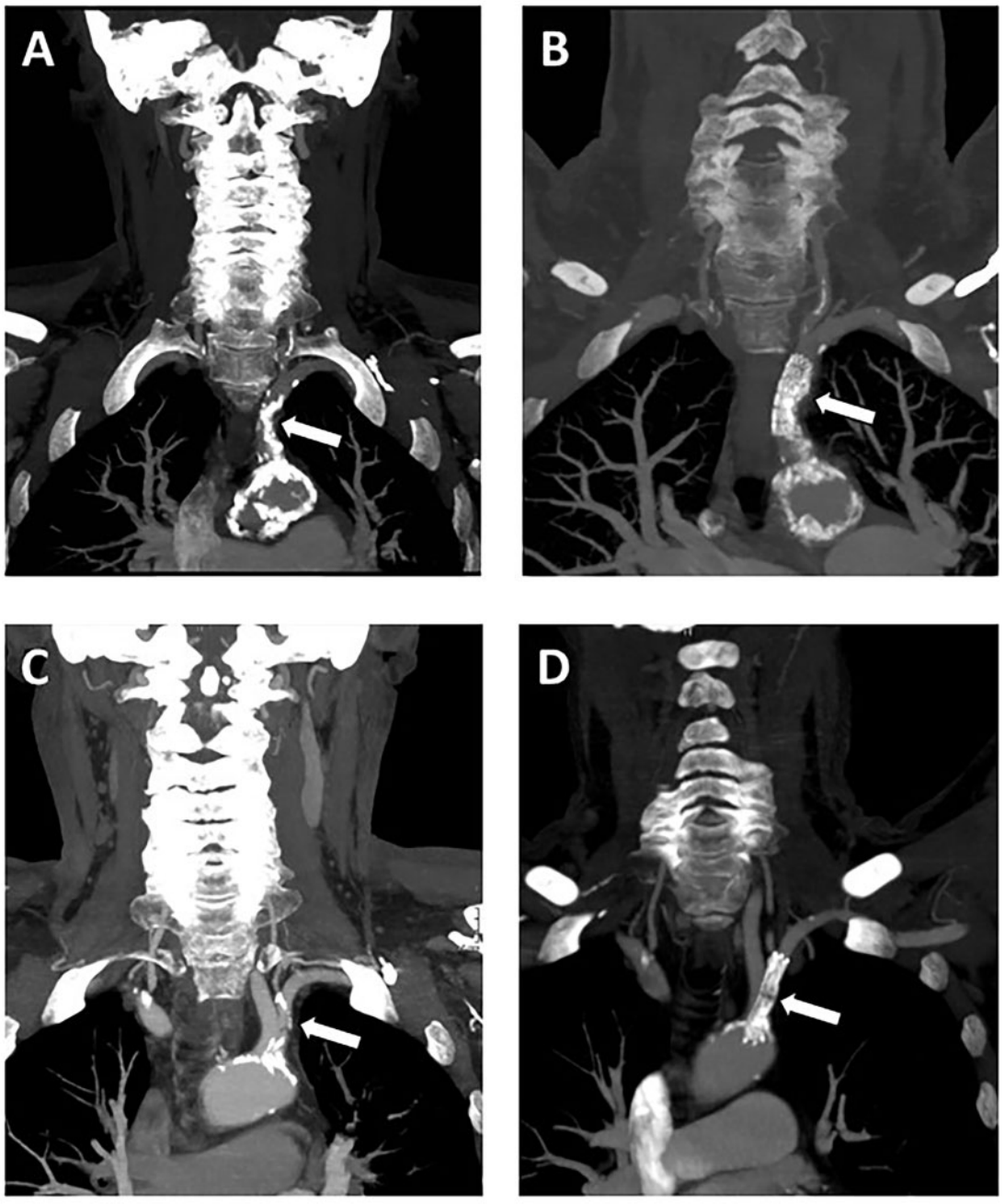


Figure 4 Chest computed tomography angiogram of Patient 1 (*A* and *B*) and Patient 3 (*C* and *D*). *A* and *C* show severe left subclavian artery stenosis before endovascular intervention, while *B* and *D* show adequate stent implantation result in both patients (arrows).

was 10 months after discharge, the patient was asymptomatic and left arm pulses were present and symmetrical.

Discussion

LIMA graft is the preferred and most frequently used conduit for myocardial revascularization.[4] During CABG, the proximal end of LIMA is normally attached to the left SA, while the distal end is anastomosed to the stenotic coronary artery. First described in 1974,[5] CSSS is defined as myocardial ischaemia resulting from the reversal blood flow from LIMA graft to left SA secondary to SA stenosis.[1] Estimated incidence of SA stenosis is 2% in the general population, and 7% in patients with peripheral artery disease.[6] However, it reaches 11.8% when CABG is performed in patients with documented peripheral artery disease.[7] All three patients in

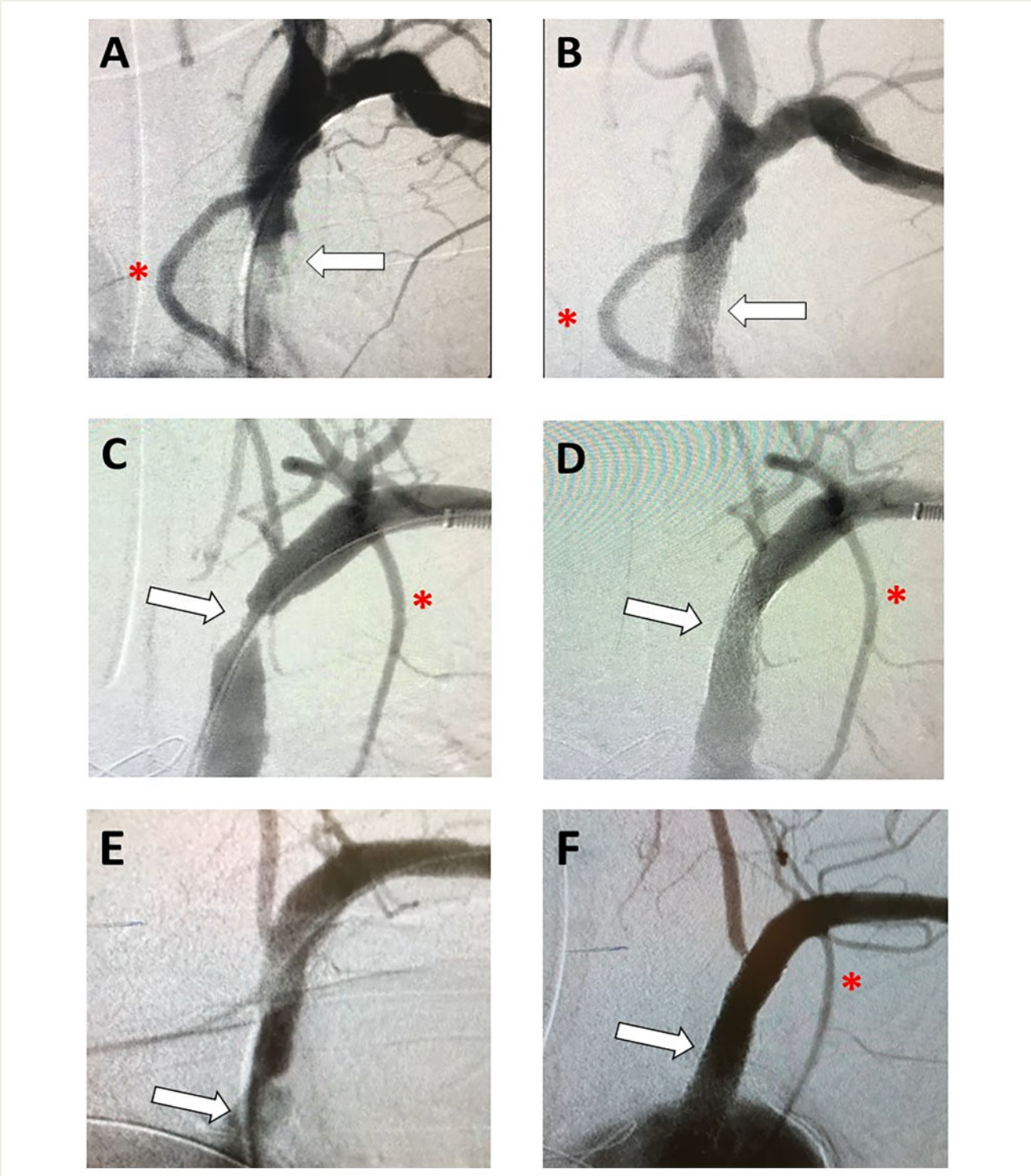
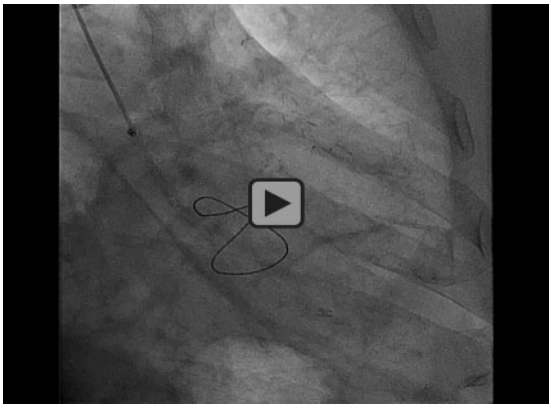


Figure 5 Left subclavian retrograde angiograms during revascularization procedures of Patient 1 (*A* and *B*), Patient 2 (*C* and *D*), and Patient 3 (*E* and *F*). *A*, *C*, and *E* show severe subclavian artery stenosis before revascularization (arrows) and *B*, *D*, and *F* document good angiographic result after stent implantation. Red asterisks mark left internal mammary artery grafts right after subclavian artery stenosis.

our series had a history of peripheral artery disease, highlighting the need for a high level of suspicion when these patients have also been surgically revascularized with an internal mammary artery bypass.



Video 3 Coronary angiography of patient 2: retrograde blood flow within left internal mammary artery graft.

As illustrated, the clinical presentation of CSSS varies widely. In asymptomatic patients, as described in case number 3, the diagnosis is very difficult and it can only be suspected by some signs detected on physical examination as an inter-arm blood pressure difference of at least 15 mmHg.[8] A Doppler ultrasound is firmly encouraged in this clinical context as it may demonstrate the presence of retrograde blood flow within the left vertebral artery.[9] Currently, a chest CT angiogram is recommended when considering the diagnosis of left SA stenosis. This imaging modality has shown high sensitivity (90%) and specificity (96%) for the diagnosis of a luminal stenosis greater than 50%.[10] Nonetheless, CSSS can induce the entire clinical spectrum of ischaemic heart disease, ranging from progressive angina (particularly associated to left arm exertion) to acute coronary syndromes,[11] as is well represented in Patients 1 and 2, respectively. According to a case series, the most frequent clinical presentation was stable angina.[12] Myocardial infarction was a much less common admission diagnosis.

In symptomatic patients studied by coronary angiography, it is important to be aware of different radiological signs that may guide to the diagnosis of CSSS. Retrograde opacification of LIMA graft should raise the suspicion of steal phenomenon,[1] as was documented in Patients 1 and 2. A difficult left SA catheterization with a pressure gradient >10 mmHg is another suggestive sign (Patient 2).[13] Left SA angiography remains the gold standard of diagnosis,[11] and it can be performed in the catheterization laboratory during coronary angiography. Myocardial perfusion scintigraphy to document myocardial ischaemia was performed in Patient 1. The role of this technique in the diagnosis of CSSS has rarely been reported.[14]

According to current guidelines,[2,3] an endovascular approach is often the default strategy for left SA stenosis. However, there are no randomized trials comparing stenting vs. surgical treatment. It seems reasonable to assume this strategy in patients with documented myocardial ischaemia secondary to SA stenosis, but this is not so clear in asymptomatic patients. In a retrospective study of 167 asymptomatic patients scheduled for LIMA-CABG, stenting for left SA stenosis was a safe strategy, with low combined incidence of death, stroke, and myocardial infarction (1.8%).[15]

Conclusions

In practice, CSSS is rarely suspected. However, patients with this syndrome may end up with serious clinical manifestations. A careful physical examination of patients with CABG, especially in high-risk populations, may provide some diagnostic clues (inter-arm blood pressure difference), that help reach the diagnosis. This case series endorses the safety and utility of endovascular treatment in CSSS and suggests a multimodality imaging approach to properly clarify this challenging diagnosis.

Lead author biography

Carlos Real is a medical doctor graduated by the University of Santiago de Compostela in Galicia, Spain. He is doing his residence in cardiology at Clínico San Carlos Hospital, located in Madrid. His main fields of interest are cardiovascular prevention and interventional cardiology.

Supplementary material

Supplementary material is available at *European Heart Journal - Case Reports* online.

Slide sets: A fully edited slide set detailing this case and suitable for local presentation is available online as Supplementary data.

Consent: The author/s confirm that informed consent for submission and publication of this case report including image(s) and associated text has been obtained from the patient in line with COPE guidance. Due to the current COVID pandemic, it was not possible to get written consent from the patients included in this study. However, witnessed verbal consent was obtained. This was discussed and agreed with the editors.

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