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CASE REPORT
Coronary intervention

Coronary-subclavian steal syndrome: a case report of a rare entity that can become a deadly threat

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Background In patients who underwent coronary artery bypass graft (CABG), the coronary-subclavian steal syndrome (CSSS) is characterized by a subclavian artery stenosis proximal to the origin of the internal mammary artery resulting in functional graft failure.

Case summary A 62-year-old gentleman underwent CABG following a non-ST elevation myocardial infarction and an angiogram showing left main stem and three-vessel disease. Forty-eight hours later he developed cardiogenic shock that improved with inotropic support and intra-aortic balloon pump insertion. However, 7 days later, he deteriorated again and even though the myocardial injury markers and echocardiogram were normal, an angiography was performed showing significant CSSS. Due to the chronic nature of his subclavian stenosis and the severity of the cardiogenic shock, the heart team decided to treated his epicardial disease percutaneously and occlude the left internal mammary artery in its mid-segment with coils. The patient was discharged home 28 days after CABG and has remained since asymptomatic with improvement in his functional class.

Discussion Coronary-subclavian steal syndrome is a rare but fatal complication with increased morbidity and mortality due to reduced awareness amongst medical professionals. Subclavian artery stenosis stenting is the gold standard treatment; herein we present a new approach for complex and very sick patients in whom it is not possible to open the subclavian artery percutaneously. Increased awareness and prompt diagnosis of this pathology in CABG patients are essential for successful outcomes.

Keywords Myocardial revascularization • Coronary artery bypass grafting • Subclavian steal syndrome • Complication • Case report

ESC Curriculum 3.2 Acute coronary syndrome • 2.2 Echocardiography • 3.4 Coronary angiography • 7.5 Cardiac surgery • 7.1 Haemodynamic instability

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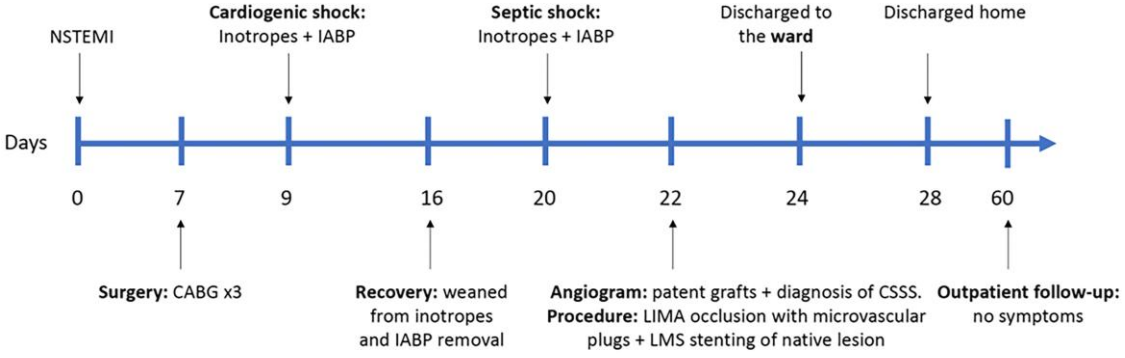
Learning points

- It is of great importance to raise awareness in basics of coronary surgery, to have protocols in place and to do basic physical evaluation of the patients.
- Coronary-subclavian steal syndrome is a rare entity, which is rarely suspected by clinicians, yet carries high morbidity and mortality if not treated promptly.
- Graft failure can present with a wide range of clinical manifestations that differ from asymptomatic to sudden cardiac death.
- Endovascular treatment of the subclavian stenosis is the gold standard treatment. However, other approaches as free left internal mammary artery (LIMA) to the aorta or even occluding the LIMA and stenting the left main stem provide also satisfactory outcomes.

Introduction

Coronary-subclavian steal syndrome (CSSS) is an uncommon complication of coronary artery bypass grafting (CABG) using the internal mammary artery (IMA).[1] In CSSS there is a haemodynamically significant proximal subclavian artery stenosis (SAS) that impedes flow down the left internal mammary artery (LIMA), hence rendering the area supplied by this graft ischaemic [in our case the left anterior descending (LAD)]. Sometimes the retrograde flow from the epicardial coronary to the subclavian, can be seen in cases where the subclavian stenosis is worse that the proximal epicardial one.[2]

Timeline



NSTEMI: non-ST elevation myocardial infarction; CABG: coronary artery bypass grafting; IABP: intra aortic balloon pump; CSSS: coronary subclavian steal síndrome; LIMA: left internal mammary artery; LMS: left main stem.

The clinical presentation of CSSS varies widely ranging from stable angina, acute coronary syndrome, sudden cardiac death or even just an incidental finding.[3]

Due to the increasing number of CABG procedures utilizing arterial grafts, particularly LIMA and right internal mammary artery (RIMA), the incidence of CSSS has also increased; however, it remains rare and is often misdiagnosed.[4] Diagnosis of CSSS remains a challenge; hence its prevalence is in general underestimated. Some series presume that CSSS ranges from 0.2% to 6.8% of patients with LIMA graft.[5]

Current guidelines are lacking recommendations for screening for SAS pre- or indeed post-CABG.[6]

Case presentation

A 62-year-old man with a past medical history of prediabetes and heavy smoker was admitted with central chest pain. At the arrival of the ambulance crew, an ECG was performed, showing noST elevation. Clinical examination showed a well perfused, normotensive patient, with no other symptoms but central chest pain. He was in sinus rhythm at 70 b.p.m. Chest auscultation revealed normal heart sounds with no added sounds or murmurs and clear chest. The patient did not have any symptoms of arm claudication in his past medical history and indeed he had good pulses in both upper limbs on clinical examination. First bloods showed a normal full blood count, with raised fibrinogen (629 mg/dL), troponin T (1038.00 ng/L), and brain natriuretic peptide (2081 pg/mL). The coronary angiogram performed via the right radial route, revealed significant left main coronary artery and three-vessel disease, and a patent right brachio-cephalic artery. The echocardiogram showed a left ventricular ejection fraction of 30% with hypokinesia in the inferior and anterior segments. As per current guideline recommendations, the carotid arteries were not imaged.[7]

In theatre, during anaesthetic induction, the patient became unstable with ST-elevation requiring an urgent initiation of surgery, precluding a BIMA procedure. Therefore, he underwent surgical revascularization using a LIMA to LAD, a saphenous venous graft (SVG) to intermediate and SVG to the posterior descending artery. He came off cardiopulmonary bypass easily, on minimal inotropic support with stable echocardiographic appearances, similar to the preoperative study. The graft assessment with the flow-meter showed patent grafts with satisfactory diastolic Doppler flows.

However, 48 h after surgery, he developed mixed cardiogenic and distributive shock requiring intra-aortic balloon pump (IABP) and high doses of inotropes. Echocardiographic assessment did not show any new regional wall motion abnormalities, and there were neither dynamic ECG changes nor troponin rise (downward trend). Both inotropic support and IABP were weaned after 7 days of treatment.

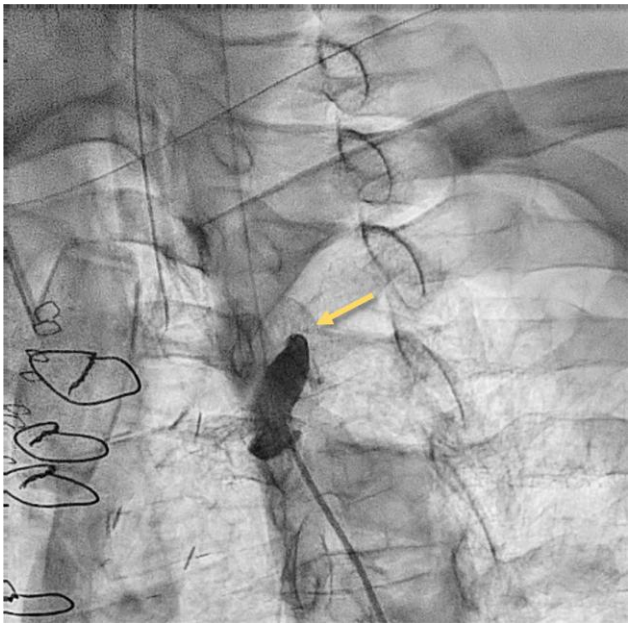


Figure 1 Selective angiography showing chronic total occlusion of the left subclavian artery (arrow).

Despite this temporal improvement, he deteriorated again after becoming septic. Inotropic support (adrenaline, noradrenaline, and milrinone) was restarted and empiric antibiotic coverage was initiated with meropenem. His repeat echocardiogram showed more profound hypokinesia in the mid-distal LAD territory, despite the absence of dynamic ECG changes and down-trending troponin values. In view of the deteriorating echocardiographic picture (suggestive of likely stunned anterior wall myocardium), he was transferred to the catheterization laboratory, where a chronic total occlusion of the left subclavian artery proximal to the origin of the LIMA was seen (*Figure 1*). The two SVGs were patent whereas the LIMA was seen filling retrogradely through the native coronary images (*Figure 2*). A peak systolic gradient

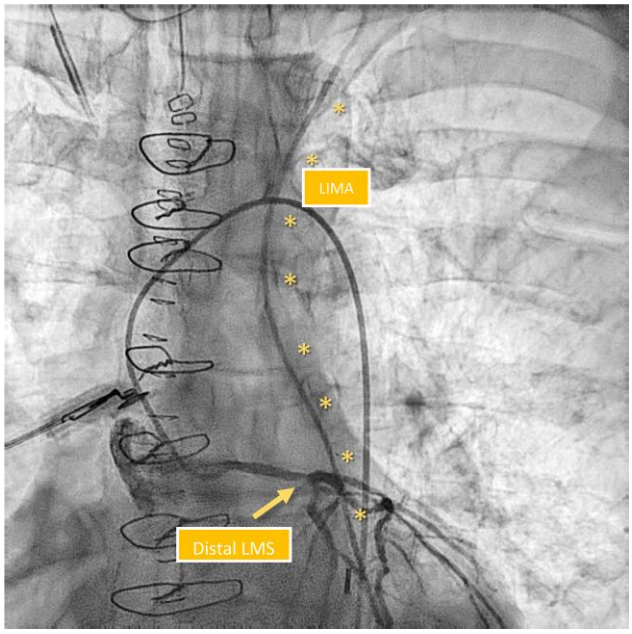


Figure 2 Coronary angiography showing severe lesion of distal left main stem (arrow) and retrograde filling of left internal mammary artery (asterisks).

of 60 mmHg between the ascending aorta and subclavian artery distal to occlusion was measured.

To improve the coronary flow down the LAD territory percutaneous coronary intervention (PCI) was performed with the implantation of a new-generation sirolimus-eluting stent in the LMS–LAD (Orsiro, Biotronik, Inc., Germany) followed by paclitaxel-eluting balloon inflation in the proximal circumflex artery (SeQuent Please, B Braun, Germany) (*Figure 3*). Additionally, a comprehensive invasive functional assessment was performed with a pressure wire to estimate the amount of blood flowing retrogradely to the left subclavian artery through the LAD. Both fractional flow reserve (FFR) and absolute flow (AF) were measured in the distal LAD with and without occlusion of the LIMA using a compliant balloon. Absolute flow was determined by continuous thermodilution during maximum hyperaemia induced by serum infusion (PressureWire × guidewire—Abbott, USA, Coroventis program —CoroFlow Cardiovascular System, Sweden, and RayFlow catheter —Hexacth Inc., France). Following PCI, a positive FFR (0.67) was obtained in the LAD with no significant differences under varying haemodynamic conditions (IABP on/off, LIMA transiently occluded, or patent). Absolute flow in proximal LAD was 0.198 L/min. However, the AF in the distal LAD was very low, but increased by 14% when the LIMA was occluded (from 0.043 to 0.050 L/min). According to these findings, we decided to occlude the LIMA using coils (Helix EV3 Concerto 2 × 6, 3 × 4, and 4 × 8 mm), as the subclavian artery occlusion was not readily treatable percutaneously and the patient's unstable condition rendered surgical re-canalization extremely hazardous. Since complete occlusion was not achieved with coils (*Figure 4*), the operators elected to also implant a Micro Vascular Plug (5.3 × 12 mm) device in the mid-LIMA (*Figure 5*).

In a more stable patient, alternative treatment options would have included reoperation and anastomosing the proximal end of the LIMA into the aorta, extra-thoracic carotid–subclavian bypass/transposition to restore the flow to the subclavian artery, or even an attempt at percutaneous re-canalization of the subclavian chronic total occlusion. However, in view of his haemodynamic instability and the presence of dual antiplatelet therapy and heparin on his therapeutic chart, the heart team agreed that a PCI was in the best interests of the patient.

Following the procedure, the patient improved significantly within days, being able to be weaned from the inotropic support and the ventilator. He was then discharged to the ward with minimal impact on his motor function from the prolonged intensive care stay.

The patient was discharged home in a good functional status 1 month after surgery following intense rehabilitation. Six months post-surgery, the patient has an EF of 40% with hypokinesia of the inferior wall, a finding present pre-surgery, but normalization of the anterior wall contractility.

Discussion

Left internal mammary artery is acknowledged as the preferred graft conduit for CABG on the LAD coronary artery. It has clearly proven its superiority thanks to its long-term patency and its chemical properties.[8] Coronary-subclavian steal syndrome has become more prevalent given the increasing numbers of CABG utilizing LIMA/RIMA conduits, yet remains a rare but serious threat.

Coronary-subclavian steal syndrome can cause no symptoms at all or be associated with acute myocardial infarction or even sudden cardiac death.[9] Of interest, the chronic left total subclavian artery occlusion did not cause any relevant symptoms to our patient pre-operatively. The syndrome became apparent only when sepsis increased the oxygen demand to the myocardium and led to a mixed cardiogenic shock clinical picture post-CABG.

Regarding the diagnosis, simple clinical assessments can help suspect this syndrome. Bilateral blood pressure measurements done in a routine manner during the preoperative evaluation can be of great value. A pressure differential of more than 20 mmHg could suggest the possibility of CSSS.[10] Ischaemia can be triggered postoperative just by exercising the upper extremities;[11] however, ideally the syndrome should be diagnosed prior to surgical intervention.

Moreover, the use of a multimodality imaging approach including Doppler ultrasound, myocardial perfusion scintigraphy, and CT is well demonstrated.[12]

Nevertheless, in urgent scenarios such as ours, probably the most useful diagnostic tool is invasive coronary angiography. Routine

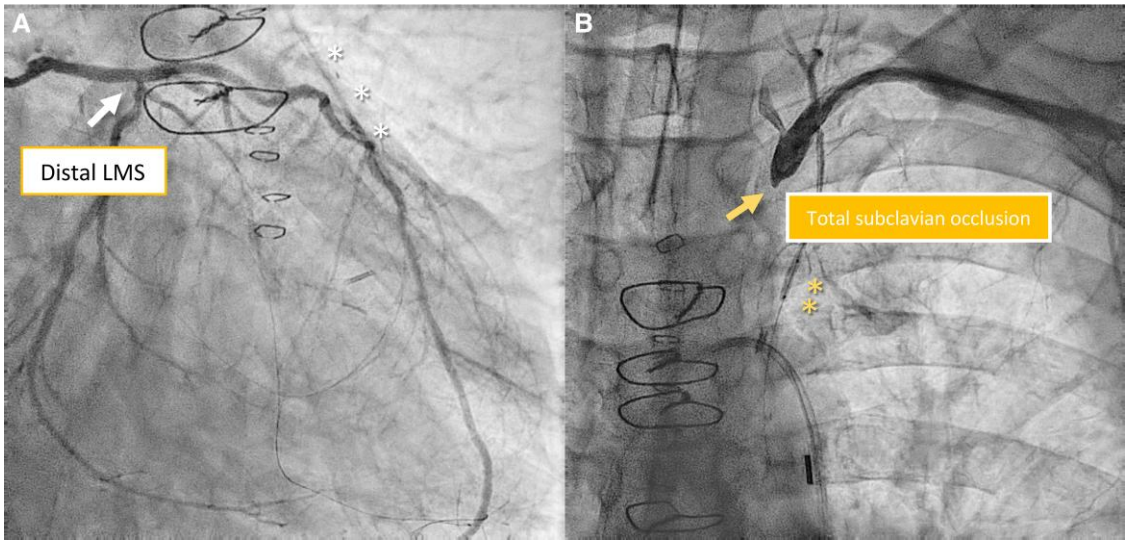


Figure 3 (*A*) Coronary angiography after percutaneous coronary intervention (white arrow). White asterisks denote left internal mammary artery retrograde filling. (*B*) Angiography of left subclavian artery distal to chronic total occlusion (arrow). Asterisks denote inflated compliant balloon for transient left internal mammary artery occlusion.

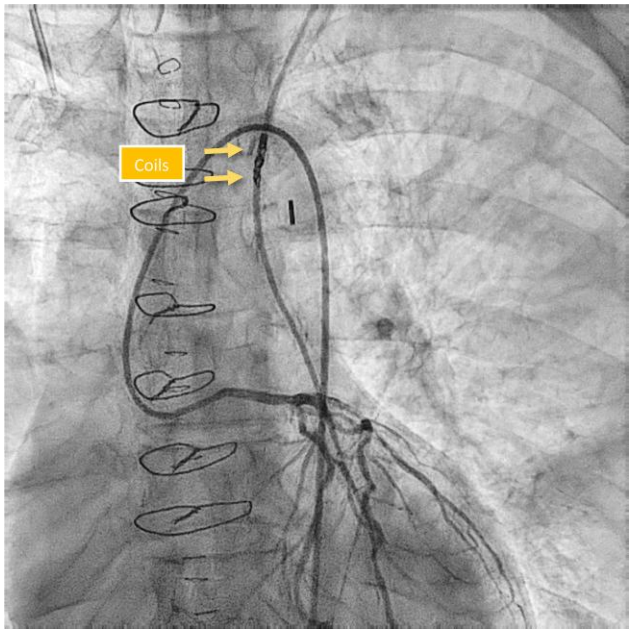


Figure 4 Coronary angiography showing patent left internal mammary artery and retrograde filling from left anterior descending after coils deployment (arrows denote coils).

subclavian artery opacification during surgical work-up using invasive or CT coronary angiography would be extremely useful to diagnose the presence of SAS and avoid this rare syndrome.[13]

In our case, the absence of significant changes in FFR with and without temporal LIMA occlusion remains unexplained; however, this may be related to a blunt hyperaemic response and haemodynamic alterations that affect the aortic (Pa) and distal (Pd) pressure measurements in a similar fashion, with and without LIMA occlusion. Of interest, however, AF, which is a direct measurement increased following the transient occlusion of the LIMA.

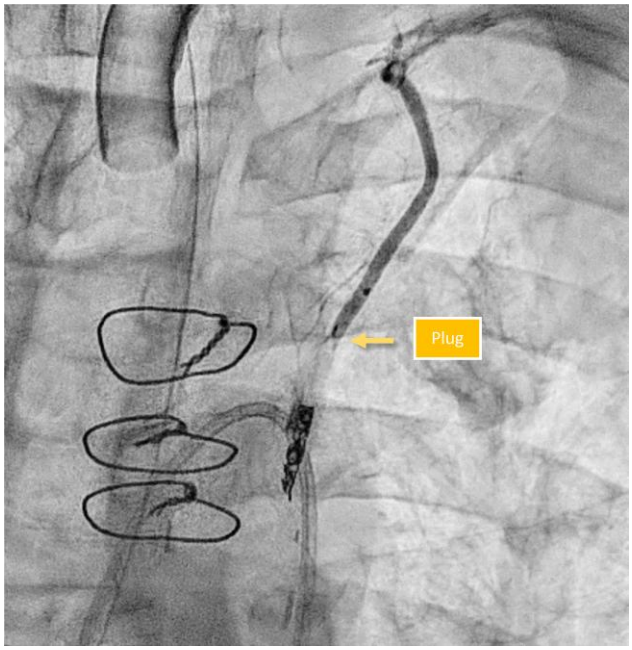


Figure 5 Complete occlusion of left internal mammary artery after plug implantation (arrow).

Coronary-subclavian steal syndrome treatment has changed a lot in the last decades, establishing the percutaneous treatment of the subclavian stenosis as the gold standard and the first line of treatment across the literature.[14]

Other approaches include cutting the LIMA and anastomosing it as a free graft to the ascending aorta. When diagnosed pre-operatively, stenting of the subclavian artery prior to CABG surgery has proven to have a low incidence of complications and in-stent restenosis.[15]

Conclusions

The diagnosis of CSSS remains a big challenge due to low levels of suspicion amongst physicians and surgeons and its variable clinical presentations. When undiagnosed and left untreated, it has the potential to become a fatal complication after IMA grafting.

To the best of our knowledge, our therapeutic approach of CSSS described in this case report, has never been described before in the literature. Even though by no means could this approach be considered a 'new gold standard', it certainly widens the treatment arsenal of this rare yet life-threatening complication.

The verdict is still out; should we routinely image subclavian arteries pre-CABG? Routine measurement of blood pressure in both upper limbs and imaging of the subclavian artery either with invasive or with CT angiography could save the patient and his treating physicians from unpleasant surprises down the line. Such an approach, however, should be balanced against the risk of complications (such as stroke) when instrumenting these arteries with catheters or indeed poor opacification and artefacts when imaged using CT.

Lead author biography

Dr Monteagudo-Vela is a consultant Cardiothoracic Surgeon in Hospital de La princesa in Madrid, Spain. Previously, after finishing her training, she spent 3 years dedicated to mechanical assist devices and Cardiothoracic Transplantation in Harefield Hospital, UK.

Supplementary material

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

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

Consent: The authors confirm that written consent for submission and publication of this case report including images and associated text has been obtained from the patient in line with COPE guidance.

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