

Coronary subclavian steal syndrome: a rare cause of acute myocardial infarction

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ABSTRACT

The coronary subclavian steal syndrome (CSSS) leading to an acute myocardial infarction (AMI) post-coronary bypass is a rare occurrence. We describe an 83-year-old Indian man who presented with AMI and was subsequently found to have CSSS. The patient had severe stenosis of his left subclavian artery ostium with retrograde flow up his left internal mammary artery graft. Angiographical steal from the left anterior descending artery was demonstrated during coronary angiogram and was thought to be the main contributing cause of his AMI. Percutaneous transfemoral angioplasty and stent implantation was performed to the left subclavian artery, with resolution of myocardial blood flow steal and anterior ischaemia.

Keywords: coronary stent, coronary subclavian steal syndrome, ischaemic heart disease, myocardial infarction, percutaneous transfemoral angioplasty

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INTRODUCTION

In the classical subclavian steal syndrome (SSS), a proximal subclavian artery stenosis is responsible for reversal of blood flow in the vertebral artery and symptoms of vertebrobasilar insufficiency that is aggravated with ipsilateral arm movement. Coronary subclavian steal syndrome (CSSS) is similarly due to a proximal subclavian artery stenosis or occlusion, and this “steals” blood away from the internal mammary artery (IMA), which is commonly used as a bypass conduit in patients with ischaemic heart disease. Increasingly, CSSS is being recognised as a rare cause for angina after coronary bypass surgery. Diversion of coronary blood flow from the left anterior descending artery (LAD) to the left arm results in angina and rarely in acute myocardial infarction (AMI). Patients with atherosclerosis and multiple coronary artery risk factors are prone to peripheral vascular disease. As the prevalence of patients undergoing coronary bypass surgery increases, invariably with a internal mammary

artery conduit, physicians need to be aware of this uncommon syndrome, and the clinical parameters for early detection.

CASE REPORT

The patient was an 83-year-old Indian man with the coronary artery risk factors of type 2 diabetes mellitus, hypertension, hyperlipidaemia and mild renal impairment. He had carcinoma of the colon, which was resected in 2001, and coronary artery bypass surgery 11 years ago in February 1995 for double vessel disease. He had a tight stenosis in the LAD and a dominant right coronary artery (RCA). The left internal mammary artery (LIMA) was grafted to the LAD and a saphenous vein conduit was grafted to the distal RCA. His left ventricular function was normal and he remained asymptomatic for many years.

The patient had recurrence of angina in 2004, nine years post-bypass surgery. A functional test for risk stratification was carried out. Sestamibi myocardial perfusion scan with intravenous dipyridamole pharmacological stress testing showed anterior lateral wall ischaemia. At that time, clinical examination revealed significantly discrepant forearm blood pressures. The right arm blood pressure was 170/90 mmHg and the left arm reading was 150/70 mmHg. The left radial pulse was weakly felt, and there was both an early systolic murmur at the left sternal edge and a bruit over the left supraclavicular area. Echocardiography demonstrated mild aortic stenosis with normal left ventricular function.

Computed tomography (CT) angiography with three-dimensional reconstruction showed extensive atherosclerotic disease with calcification over the aortic arch and the proximal portion of the great vessels especially at the origin of the left subclavian artery (Fig. 1). Cardiac catheterisation showed native triple vessel disease and a tight 90% stenosis of the left subclavian artery. Both bypass grafts were patent. Since the patient was asymptomatic for the left subclavian stenosis and he had mild renal impairment, the decision was to treat the circumflex artery stenosis for which he underwent uneventful angioplasty and stenting in January 2004. The left subclavian artery stenosis was then managed expectantly.

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Fig. 1 CT angiogram (maximum intensity projection) of the aortic arch shows extensive arterial calcification with a calcified plaque obscuring the stenosis at the origin of the left subclavian artery (arrow).

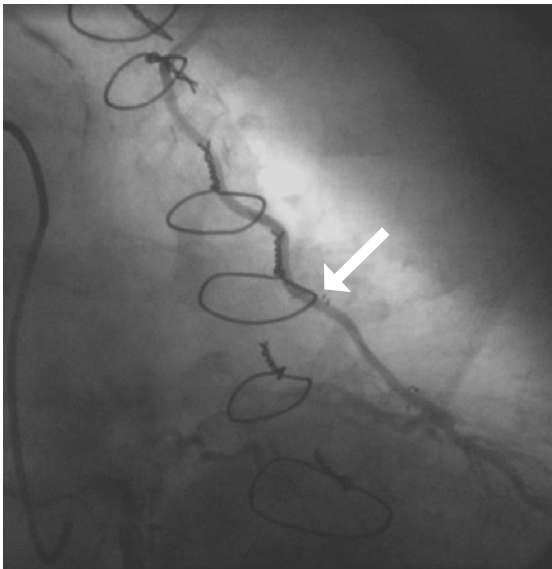


Fig. 2 Coronary angiogram shows retrograde blood flow from the LAD to the subclavian artery via the LIMA (arrow).

The patient remained stable on follow-up for the next two years until December 2005 when he was admitted for AMI. He had started to experience intermittent rest angina, which was aggravated with use of his left arm. The episode of worst pain that brought him to hospital was when he was reaching up to tidy his cupboard. He did not have any symptoms of dizziness or syncope. Clinical examination showed marked discrepancy in his forearm blood pressures. His right arm blood pressure was 180/90 mmHg and his left arm blood pressure was 100/70 mmHg. The left radial pulse was absent and the left brachial pulse was weak.

His electrocardiogram (ECG) showed a sinus rhythm and preexisting right bundle branch block morphology. Serial cardiac enzymes showed a significant rise in the creatinine kinase and Troponin T fractions. The diagnosis of a non-ST elevation myocardial infarction

(NSTEMI) was made based on the chest pain history, cardiac enzyme elevation and lack of ST elevation on the ECG. The patient had coronary catheterisation done two days after the NSTEMI, which showed that both his bypass grafts were patent and his prior stented segment in the circumflex artery had only minor disease. Retrograde myocardial blood flow was demonstrated, with a selective injection of contrast agent in the native LAD via the patent LIMA to near the ostium of his left subclavian artery. (Fig. 2). There was a step-down gradient of 80 mmHg across the tight 95% stenosis at the ostium of the left subclavian artery (Fig. 3).

The patient was diagnosed as having AMI secondary to CSSS arising from severe stenosis of his left subclavian artery as the main contributing factor. A staged angioplasty for the culprit subclavian artery was carried out to decrease the contrast load. Retrograde approach via the left brachial artery was attempted but was unsuccessful. Access was achieved from the right groin. A 7F JL 4 cm short tip guider was inserted, anticoagulation was achieved with 6,000 units of intravenous heparin. The tight left subclavian artery was crossed with a Terumo 0.035 inch diameter hydrophilic-coated wire and directly stented with an Express LD 10mm by 25 mm balloon expandable stent at 10 atmospheres. The step-down gradient was lost and coronary catheterisation showed no further retrograde flow from the LAD to LIMA (Fig. 4). The patient has since remained well on follow-up, with no further episodes of angina and forearm blood pressures have also equalised.

DISCUSSION

This patient had an unusual presentation of a rare clinical syndrome. CSSS may give rise to angina but rarely cause an AMI. For coronary subclavian steal to occur, the patient will need to have both a patent LIMA as well as severe disease in the left subclavian artery. CSSS should not be confused with SSS, which results from retrograde blood flow from the ipsilateral vertebral artery due to proximal subclavian artery stenosis. SSS was first postulated by Harrison⁽¹⁾ in 1829 but was only angiographically demonstrated by Contorni⁽²⁾ in 1960, and the syndrome was coined in 1961 by Reivich et al⁽³⁾. SSS typically results in ipsilateral vertebral basilar symptoms like dizziness, vertigo, ataxia and syncope.

It was not until the late 1970s that CSSS was increasingly recognised as a cause of angina post-bypass surgery and up till the early 1990s, only a handful of cases was reported⁽⁴⁻⁶⁾. The reported incidence in surgical series ranges from 0.5%–2%⁽⁷⁻⁹⁾. The increasing documentation of this phenomenon and its potentially catastrophic consequences in recent studies suggests that the incidence of the problem has been under-reported and that its clinical impact has been underestimated⁽¹⁰⁾.

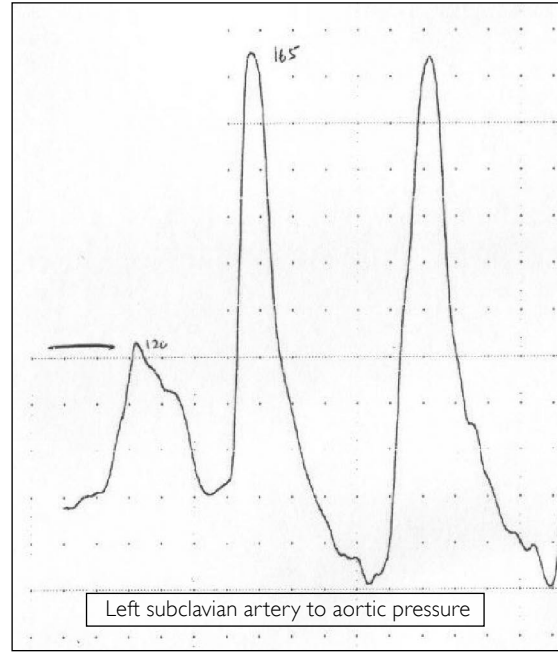


Fig. 3 Coronary angiogram shows a critical stenosis at left subclavian artery ostium (arrow) with a large step-down gradient.

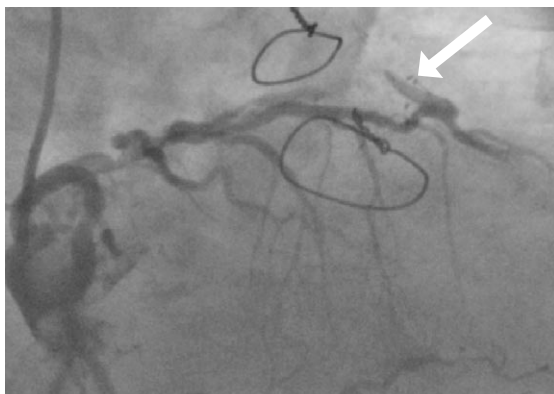


Fig. 4 Post-stenting coronary angiograms show loss of retrograde flow up the LIMA (arrow).

The aetiology for CSSS is invariably atherosclerosis, and the risk of associated peripheral vascular disease in patients with coronary artery disease is well established. Rarely, Takayasu's arteritis as the cause has been described in case reports^(11,12). The rarity of this syndrome is probably due to the fact that it is localised to the left, as the LIMA rather than right IMA is the conduit of choice in cardiac revascularisation and in addition, the site of stenosis of the subclavian artery must be proximal to the LIMA origin. In some patients, both SSS and CSSS can occur with a combination of neurological complaints and angina secondary to occlusion of the subclavian artery^(13,14). Some authors coined the term "coronary-subclavian-vertebral steal syndrome" for this combination syndrome⁽¹⁴⁾.

Clinically significant stenosis will produce a differential forearm blood pressure of 20 mmHg⁽¹⁵⁾, and this should be routinely sought for in the outpatient setting, especially for patients primed for bypass operation^(7,8). Significant lesions missed before the LIMA is grafted can lead to potentially catastrophic postoperative complications⁽⁸⁾. Bruits over the supraclavicular area may be present and other associated bruits over the carotids, abdomen and groin should be looked for as clues for peripheral vascular disease elsewhere. Doppler spectral technique is a valuable first line tool for detection of a haemodynamically significant stenosis of the left subclavian artery. Imaging with CT angiogram or contrast-enhanced magnetic resonance imaging can be considered but cardiac catheterisation remains the gold standard for diagnosis. During the procedure, direct measurement of the pressure gradient can also be obtained with

concomitant demonstration of myocardium blood flow steal via the LIMA.

Carotid-subclavian bypass grafting was the procedure of choice for management of the CSSS in the 1970s and 1980s^(16,17). This has been surpassed by percutaneous angioplasty⁽¹⁸⁾ and stenting of the subclavian artery⁽¹⁹⁻²¹⁾. For total occlusions of the subclavian artery not amenable to endovascular strategies, surgical options with carotid-subclavian bypass or subclavian-carotid transposition provide excellent means with good mid- and long-term results^(17,22). In summary, with the increasing use of the IMA in the coronary artery bypass graft procedure, this uncommon syndrome will be encountered more frequently in the future. Looking out for discrepant forearm blood pressures of more than 20 mmHg is essential if CSSS is suspected. Treatment with percutaneous angioplasty and stenting is an accepted modality with good short- and mid-term results.

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