Case report

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A case report of probable severe coronary artery spasm (initially treated with unnecessary coronary artery bypass surgery) and subsequent coronary subclavian steal syndrome

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Coronary artery bypass graft surgery with a left internal mammary artery graft to the left anterior descending artery (LAD) was performed on a patient with severe ostial LAD stenosis, which was subsequently found to be a coronary artery spasm. This patient was later found to have a steal phenomenon from LAD to the subclavian artery via the internal mammary graft because of severe ostial subclavian artery stenosis, the so-called coronary subclavian steal syndrome. The patient's angina completely resolved once the subclavian stenosis was treated with percutaneous balloon angioplasty and stenting.

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Case report

A 57-year-old lady first presented with chest pain at rest lasting for 1 h in 2000. Her background history included hypertension, hypercholesterolaemia, peripheral vascular disease and heavy smoking. There was anterior T-wave inversion on the ECG. Due to the ongoing chest pain and ECG changes, she was transferred to a tertiary cardiac centre where coronary angiography showed a severe ostial left anterior descending (LAD) stenosis with unobstructed left main stem artery (LMS), left circumflex (LCX) (Fig. 1) and right coronary arteries (RCA). She underwent an urgent off-pump coronary artery bypass graft (CABG) with a left internal mammary artery (LIMA) to LAD. She was pain-free and was discharged after a few days. In 2003, she underwent uneventful bilateral common iliac angioplasties and subsequently required aortobifemoral bypass in 2004.

The patient was reviewed in the cardiology clinic subsequently with chest pain in April 2005. A thallium myocardial perfusion scan showed normal myocardial perfusion and the left ventricular gated study showed mild left ventricular systolic impairment. Echocardiography showed mild global left ventricular dysfunction, with no regional wall motion abnormalities. She was reassured and her symptoms settled at that stage.

She presented with typical history of angina for 2 months in July 2009 and underwent an exercise treadmill test, during which she developed chest tightness and 1 mm ST-depression in V3–V5. Her coronary angiogram now showed the ostium of the LAD to be normal (Fig. 2) with no evidence of atheroma. Once again, the LMS, LCX and RCA were normal. The LIMA graft retrogradely filled from the native LAD and spilt over into the left subclavian artery (Fig. 3). It was difficult to intubate the LIMA at its ostium, as the catheter would not pass through what appeared to be a very severe ostial left subclavian stenosis (Fig. 4). Computed tomography aortogram was arranged which demonstrated a short segment, significant stenosis at the origin of the left subclavian artery, proximal to the origins of the left vertebral artery and the LIMA (Fig. 5), and again the LIMA graft appeared patent. A diagnosis of coronary subclavian steal syndrome (CSSS) was made. The subclavian stenosis was treated with percutaneous balloon angioplasty and an 8×18 mm Cordis balloon-mounted stent (Cordis Corporation, USA) was deployed with excellent result (Fig. 6). There was complete resolution of symptoms after this procedure. AQ1

Discussion

We reported an unusual case in which not only coronary artery bypass surgery was performed for probable coronary spasm, but also there was subsequently symptomatic CSSS exacerbated by the high flow through an unobstructed LAD.

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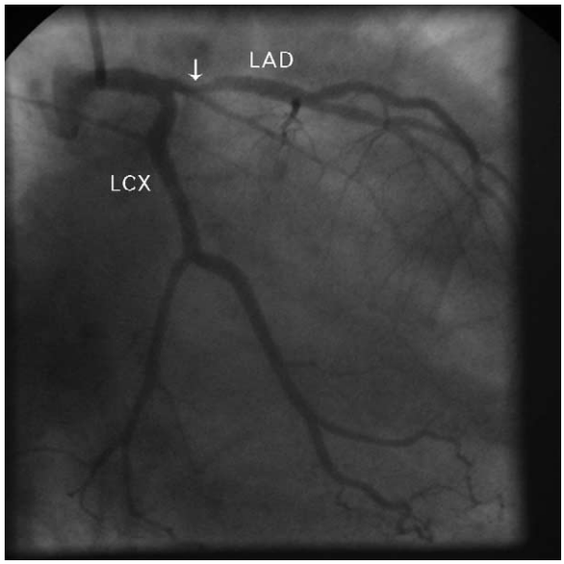
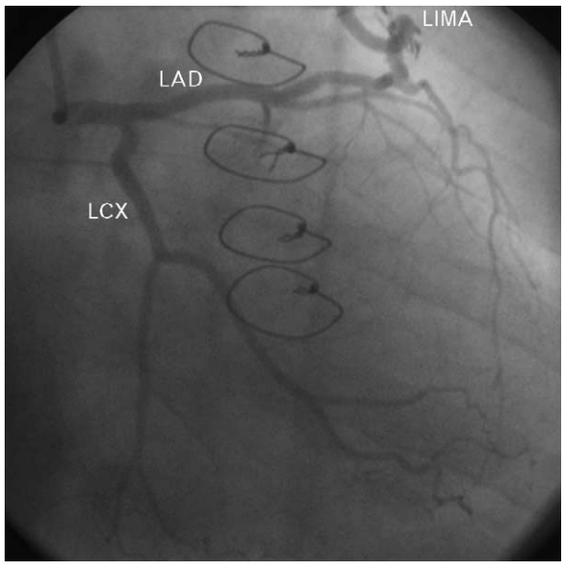
Fig. 1



Right anterior oblique view of left coronary system in 2000 showing severe stenosis in the proximal left anterior descending artery (white arrow). LAD, left anterior descending artery; LCX, left circumflex artery.

In retrospect, the original angiographic appearance of the ostial LAD was probably due to severe spasm of the LAD. The other theoretical explanations for the angiographic changes in the proximal LAD would be coronary artery remodelling or atherosclerotic plaque regression.

Fig. 2



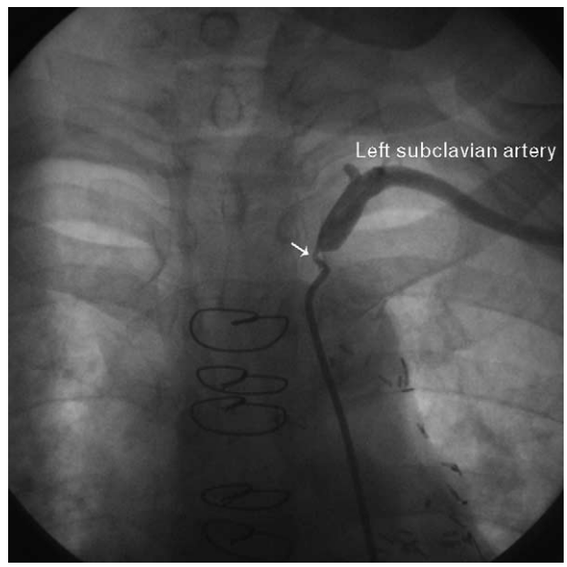
Right anterior oblique view of left coronary system in 2009 showing no stenosis in the left anterior descending artery. LAD, left anterior descending artery; LCX, left circumflex artery; LIMA, left internal mammary artery.

Fig. 3

Angiogram showing retrograde flow from left internal mammary artery graft to the subclavian artery. LIMA, left internal mammary artery.

Both of these entities have been documented in those lesions which have 40–50% stenosis, but not in more severe lesions. A study by Hartmann *et al.* [1] showed that lesions with baseline plaque burden more than 40% may continue to develop positive remodelling leading to

Fig. 4



Angiogram showing severe stenosis (white arrow) at the origin of the subclavian artery.

Fig. 5

Computed tomography angiogram (coronal reconstruction) showing severe stenosis at the origin of subclavian artery.

compensatory enlargement of the lumen, but no studies have proven these entities in lesions which have severe stenosis, as found in our case. Also, our patient was only on a standard dose of HMG-CoA reductase inhibitor (simvastatin 40 mg), which would not account for significant plaque regression. Therefore, we believe that the most likely diagnosis in this situation is coronary spasm, although there may be underlying plaque at the site of the lesion. Intracoronary nitrate is not routinely used in diagnostic angiography when spasm is not suspected, though it clearly would have helped differentiate the stenosis from spasm in this case.

Fig. 6

Angiogram postleft subclavian artery stent showing no residual stenosis.

Coronary artery spasm (CAS) has been shown to play an important role in the pathogenesis of angina and myocardial infarction, with or without coronary artery stenosis [2]. In patients with CAS, calcium channel blockers relax the coronary smooth muscles and produce coronary vasodilatation. The decrease in frequency of vasospastic-type 'Prinzmetal angina' is attributed to the widespread use of these drugs. Long-acting nitrates were also found to be efficacious, and the vasodilatory effect may be additive to calcium antagonists. Medically intractable life-threatening coronary spasm has been treated successfully with internal mammary graft in two patients, despite angiographically normal coronary arteries [3].

CSSS is an uncommon complication of CABG using LIMA with a reported incidence varying between 0.07 and 3.4% [4,5]. The cause of CSSS is invariably due to atherosclerosis, and the risk of associated peripheral vascular disease in patients with coronary artery disease is well established. Symptoms are reported to occur between 2 and 31 years following CABG surgery. Symptoms presenting within a year of CABG usually suggest a subclavian stenotic lesion missed at surgery [6]. Our patient did not have any stenosis of the LAD and, therefore, the flow in the LAD–LIMA–subclavian artery was brisk, increasing the likelihood of a steal phenomenon. This is different from other reported cases of this condition wherein most of the patients had LIMA graft for severe stenosis of the LAD.

The treatment options for CSSS include surgical and radiological-guided endovascular procedures, depending on the anatomy and severity of the lesion. The advantages of radiological procedures are minimal invasiveness, avoidance of general anaesthesia, less morbidity and mortality along with excellent short-term results [7]. Carotid-subclavian bypass graft with a synthetic conduit was reported to have a primary patency rate of 92% at 10 years [8]. As far as we are aware, there has been no prospective randomized trial which directly compares stenting with surgery in subclavian artery stenosis. A retrospective analysis from a single institute, however, showed superior 5-year patency rate with carotid-subclavian bypass graft surgery than percutaneous transluminal angioplasty/stenting in subclavian artery disease (96 vs. 70%. $P < 0.0001$) [9]. AQ2

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