Myocardial ischaemia due to subclavian stenosis after coronary artery bypass graft: a case report

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Introduction
Subclavian artery stenosis occurs up to 4.6% in patients who are referred for a coronary artery bypass graft (CABG). Subclavian artery stenosis can compromise the blood flow in the ipsilateral mammary artery.

Case presentation
In this case report, we describe a patient with prior history of CABG and peripheral vascular disease, who presented with recurrent chest pain symptoms. Cardiac perfusion imaging using Rubidium-82 positron emission tomography showed extensive ischaemia in the anterior wall. Coronary angiography showed an ipsilateral (left) severe subclavian stenosis, while there was no significant stenosis in the bypass grafts. Patient’s symptoms resolved after percutaneous intervention of the left subclavian artery.

Discussion
The presence of subclavian artery stenosis can result in myocardial ischaemia after prior CABG utilizing the internal mammary artery. A history of peripheral vascular disease and a blood pressure difference between the upper extremities >15 mmHg are clinical predictors of subclavian artery stenosis. Percutaneous angioplasty and stenting is considered the first-line treatment for subclavian artery stenosis. Surgical management should be considered after failure of endovascular treatment in low-surgical-risk patients.

Keywords
Case report • Ischaemia • Subclavian stenosis • Coronary artery bypass graft

Learning points
• Myocardial ischaemia due to subclavian artery stenosis is an important alternative diagnosis to coronary artery disease after coronary artery bypass graft.
• A history of peripheral vascular disease and a blood pressure difference between the upper extremities >15 mmHg are clinical predictors of subclavian artery stenosis.
• First-line treatment for subclavian stenosis is percutaneous revascularization.

Introduction
Subclavian artery stenosis occurs up to 4.6% in patients who are referred for a coronary artery bypass graft (CABG).1,2 Subclavian artery stenosis can cause lower pressure distal from the stenosis, compromising blood flow in the ipsilateral mammary artery. In severe left subclavian artery stenosis, upper extremity exertion can result in a coronary subclavian steal syndrome (CSSS) due to retrograde blood flow up the left internal mammary artery (LIMA). After CABG, this condition may result in myocardial ischaemia despite patency of the grafted vessels. Common risk factor for subclavian stenosis is atherosclerosis, while less common causes are large vessel arteritis (i.e. Takayasu disease, giant cell arteritis), fibromuscular dysplasia, radiotherapy, thoracic outlet syndromes, or congenital malformations of the right aortic arch.3–5 We report a case of a patient with previous history of a LIMA to left anterior descending artery (LAD) bypass graft who reported angina. After detection of severe ischaemia on Rubidium-82 positron emission tomography (Rb-82-PET), coronary angiography revealed subclavian artery stenosis as the cause of myocardial ischaemia. Soon afterwards an out-of-hospital cardiac arrest occurred and an emergency coronary angiography was performed.

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A 68-year-old man with a history of coronary artery disease reported stable angina and exertional dyspnoea. Medical history revealed hypertension, endovascular treatment of lower extremity peripheral vascular disease (percutaneous transluminal angioplasty of the left superficial femoral artery), and a two-vessel CABG including a LIMA to the LAD and venous graft aorta-obtuse marginal (aorta-OM) and a percutaneous coronary intervention (PCI) of the left main stem. No hyperlipidaemia, diabetes, or smoking was reported. On physical examination blood pressure was 166/85 mmHg (no bilateral arm measurement was performed), heart rate 90/min. Resting ECG shows signs of left ventricle hypertrophy with strain and ST segment elevation in V1–V3. Echocardiography showed dilated left ventricle with hypokinesia of the anterior wall, left ventricular ejection fraction (LVEF) 25%. To determine myocardial perfusion, non-invasive imaging using Rubidium-82 PET was performed, demonstrating extensive ischaemia in the anterior wall (myocardial territory supplied by the LAD) (Figure 1) and decreased LVEF (20% after Regadenoson and 23% in rest). Following the results of the Rubidium-82 PET, the patient was scheduled for a coronary angiography. An emergency coronary angiography was performed after the patient presented with an out-of-hospital cardiac arrest. Coronary angiography via the right femoral artery was performed showing no stenosis in the LIMA or other coronary arteries (Figure 2). However, selective arteriography of the left subclavian artery demonstrated a plaque that is hazy, ulcerated, and causes a severe stenosis of the proximal part of left subclavian artery. Percutaneous revascularization was performed successfully using an 8.0×39 mm self-expandable bare metal stent (Figure 4). Predilatation was performed using serial balloon inflations of 10 mm and 12 mm. Treatment of the subclavian artery stenosis was enough to restore antegrade perfusion of the LAD territory via the LIMA graft.

After 2-years’ follow-up, patient reported again atypical chest pain and fatigue. Follow-up Rubidium-82 PET was performed (2 years after percutaneous intervention of the left subclavian artery) showing no myocardial ischaemia (Figure 5) and normalization of left ventricle systolic function (57% after Regadenoson and 52% in rest).

**Discussion**

We described a case of angina in a post-CABG patient with symptomatic subclavian artery occlusive disease. Although the most common cause of angina in a post-CABG patient is progression of native vessel coronary atherosclerosis and disease in the grafts, a subclavian stenosis should be considered. Patients with atherosclerotic occlusive plaques in the subclavian artery are usually asymptomatic because of the abundant collateral blood supply in the head, neck, and shoulder. Coronary subclavian steal syndrome has a reported incidence of approximately 2.5%.

The classical presentation of CSSS is angina induced by left upper extremity activity. A severe stenosis can also result in reversal of blood flow in the vertebral artery causing posterior circulation vertebro-basilar symptoms (like dizziness or syncope) also known as subclavian vertebral steal syndrome. The case presented here issues two clinically important points. Firstly, we did not determine the pressure difference between the right and left arm in follow-up. Secondly, identifying the risk of subclavian stenosis was important, especially in a patient with a history of peripheral vascular disease. A history of peripheral vascular disease and a blood pressure difference between the upper extremities greater than 15 mmHg are clinical predictors of subclavian artery stenosis.

However, bilateral blood pressure measurement can still miss patients who have equal bilateral subclavian artery stenosis. The benefits of routine screening before CABG are not known. Radionuclide imaging is included in the follow-up strategies after PCI and CABG in patients with symptoms. In our case, we showed ischaemia in the anterior wall on Rubidium-82 PET (indicating decreased flow in the LIMA-LAD); however, the true mechanism of this decreased coronary blood flow in stress is not visualized using this imaging technique. Selective arteriography of the left subclavian artery demonstrated a plaque that is hazy, ulcerated, and causes a severe stenosis of the proximal part of left subclavian artery.

Other non-invasive imaging modalities, such as Doppler ultrasound, computed tomography, and magnetic resonance angiography could be performed to identify a subclavian artery stenosis. Current treatment options include a surgical bypass, percutaneous angioplasty, and stenting. Percutaneous angioplasty with stent support is considered the first-line treatment for subclavian artery stenosis. A surgical method should be considered after failure of endovascular treatment in low-surgical-risk patients. The patency rates of subclavian arteries after percutaneous revascularization are high (75%) at 10 years’ follow-up.

**Conclusion**

Coronary ischaemia due to subclavian artery stenosis is an important alternative diagnosis to coronary artery disease after CABG. Stenosis of the left subclavian artery can result in decreased flow in the ipsilateral internal mammary artery, compromising coronary circulation supplied by a LIMA graft.
Figure 1 Rubidium-82 positron emission tomography. The stress study demonstrates a decreased perfusion of the anterior wall, while the rest study showed normal perfusion, indicative of myocardial ischaemia of the left anterior descending artery territory.
Consent: The authors confirm that written 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.

Conflict of interest: none declared.

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Figure 2
Angiography demonstrates normal patent grafts (left internal mammary artery-left anterior descending artery, aorta-obtuse marginal).

Figure 3
Angiography demonstrates a plaque that is hazy, ulcerated and causes a stenosis of the proximal left subclavian artery (white arrow).

Figure 4
Angiography after revascularization with bare metal stent showing restoration of normal flow in the subclavian artery.
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Figure 5 Rubidium-82 positron emission tomography. Two-years after bare metal stent of subclavian stenosis demonstrating no myocardial ischaemia.
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