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Case Report

CASE REPORT

Pseudo-hypotension with acute pulmonary oedema due to simultaneous bilateral subclavian artery stenosis in a patient with coronary artery bypass graft surgery using bilateral internal mammary arteries: a case report

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Abstract

A 75-year-old woman, with a history of bilateral internal mammary artery–coronary artery bypass graft surgery, developed hypotension and pulmonary oedema posing as cardiogenic shock. Severe bilateral subclavian artery stenosis emerged to be the cause of ischaemic myocardial dysfunction and heart failure. An emergency endovascular treatment was successfully performed. The presence of simultaneous bilateral subclavian artery narrowing as the pathophysiologic mechanism of myocardial ischaemia makes this case remarkable.

INTRODUCTION

Subclavian artery disease is an under-recognized peripheral artery disease that may present as arm claudication or vertebrobasilar insufficiency, more commonly known as subclavian steal syndrome [1–3]. In addition, following left internal mammary artery (LIMA)–coronary artery bypass surgery, some patients develop ipsilateral subclavian artery stenosis (SAS), causing myocardial ischaemia that requires endovascular treatment (EVT) [2–7]. Other patients may require revascularization for SAS before a LIMA–coronary artery bypass surgery [8]. Here, we present a case of simultaneous bilateral SAS causing severe myocardial dysfunction after bilateral IMA–coronary artery bypass grafting.

CASE PRESENTATION

A 75-year-old woman presented to our emergency room complaining of severe dyspnoea. She had undergone coronary artery bypass graft surgeries (CABGs) 8 and 13 years prior to the current admission. LIMA was grafted to the left anterior descending (LAD) artery, the right IMA (RIMA) to the obtuse marginal branch and the saphenous vein graft to the distal left circumflex artery. The patient had hypertension, diabetes mellitus and hyperlipidaemia. Her blood pressure (BP) was stable at about 110–130/60–70 mmHg with medications. Further, her blood sugar and lipid levels were under control: HbA1c of 6.5%–6.7% and LDL cholesterol of 100 mg/dL. She enjoyed regular physical activities such as walking. Bilateral neck bruit was observed 2 years prior to

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her admission. Magnetic resonance (MR) angiography revealed less than 50% stenosis of the bilateral carotid arteries and 50% stenosis of the bilateral subclavian arteries. Ankle–brachial index (ABI) was 1.26 for the right side and 1.42 for the left side (127/63 mmHg in the right arm, 114/60 mmHg in the left arm, 160/61 mmHg in the right leg and 162/64 mmHg in the left leg). ABI increased to 1.62 on the right side (113/68 and 184/71 mmHg in the right arm and leg, respectively) and 1.62 on the left side (118/69 and 191/77 mmHg in the left arm and leg, respectively) 1 year prior to admission. There was no inter-arm BP difference and no sign of arm claudication. Therefore, arm perfusion was considered normal. Elevation of ABI was attributed to artery stiffness at this time.

The patient had been asymptomatic until 6 months prior to admission when she started experiencing chest discomfort on exertion. Her BP measured in the right arm at that time was 126/70 mmHg. Her symptoms started worsening 2 months prior to admission, and her BP dropped to 94/60 mmHg. A 12-lead electrocardiogram (ECG) revealed worsening of the ST segment depression and widening of the QRS interval, suggesting non-specific intraventricular conduction delay. Echocardiography showed decreased left ventricular wall motion with an ejection fraction (EF) of 40% (previous EF was >55%). Pimobendan (2.5 mg bid; a cardiotonic) was administered, and her condition slightly improved. Her BP in the right and left arms was 70/40 and 80/50 mmHg, respectively, 1 week before admission. At this point, BP medication was discontinued.

The patient was scheduled for cardiac catheterization and angiographic evaluation of the bypass grafts and native coronary arteries. However, before the scheduled admission date, she developed severe dyspnoea and was taken to the emergency room. The patient exhibited the following vitals: BP was difficult to measure in both arms; respiratory rate was 30/min; heart rate was 86/min and body temperature was 36.0°C. Chest X-ray revealed severe bilateral pulmonary oedema, and an ECG revealed diffuse ST segment depression with non-specific intraventricular conduction delay. Cardiac troponin I was found to be positive, and initial creatinine phosphokinase level was 66 U/L that increased to 720 U/L (normal range: 41~140 U/L). Kidney and liver functions were normal. The patient was intubated, taken to the intensive care unit and started on intravenous dobutamine and furosemide. While she continued to be hypotensive despite vigorous treatment, urine output was well maintained (950 mL in the first 4 h and 50–100 mL/h thereafter), and dyspnoea quickly improved. Although systolic BP was low (around 60–70 mmHg in both arms), her femoral artery was bilaterally well palpable.

DIFFERENTIAL DIAGNOSIS

Due to the persistent hypotension observed in this patient, cardiogenic shock was initially suspected. However, an adequate urine output, rapidly improving dyspnoea and the absence of metabolic acidosis were not consistent with the usual symptoms of cardiogenic shock. In addition, there was a bruit in the bilateral subclavian areas and the neck. As a result, urgent cardiac catheterization was performed. Bilateral SAS was suspected to be the cause of pseudo-hypotension, diffuse myocardial dysfunction and acute pulmonary oedema. Simultaneous invasive BP was measured in the bilateral brachial arteries and aorta. Aortic pressure was normal at 130/70 mmHg (mean, 90 mmHg), whereas pressures in the left and right brachial arteries were 78/52 (mean, 61 mmHg) and 65/50 mmHg (mean, 55 mmHg), respectively (Fig. 1A). Angiographic evaluation showed severe

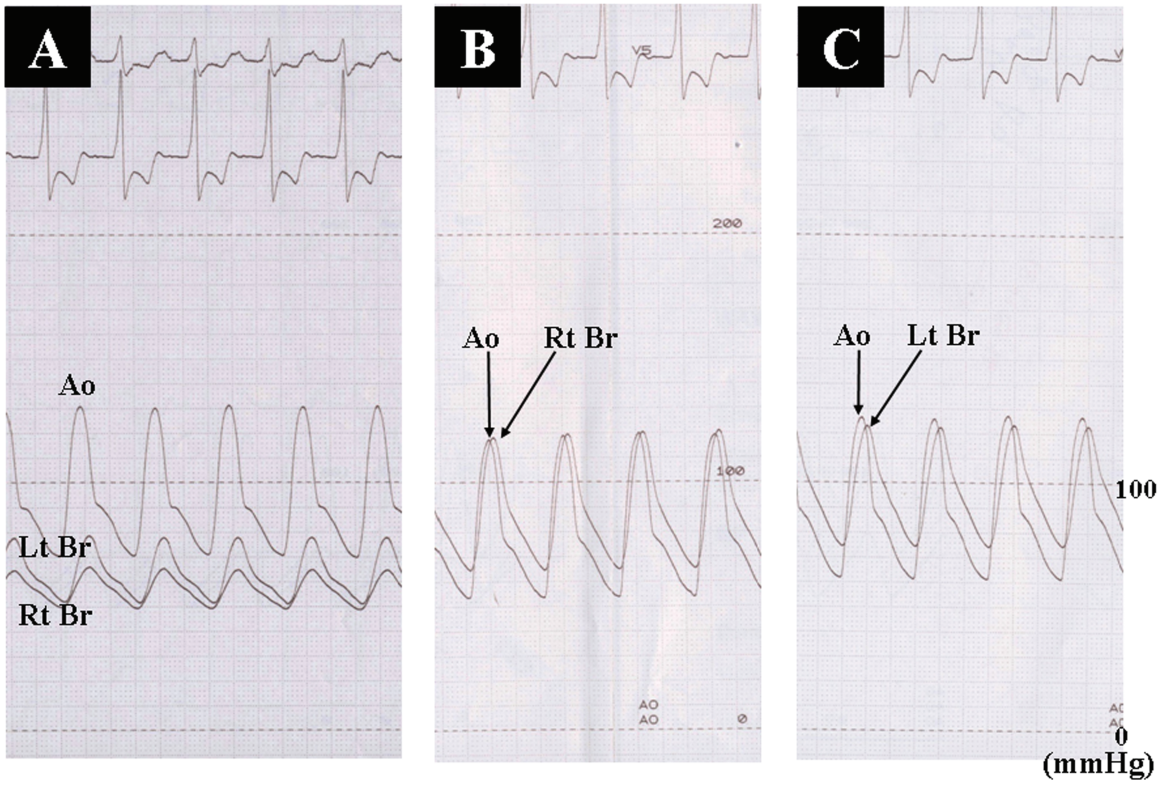

Figure 1: At baseline, pressure gradients were marked between the aorta and bilateral brachial arteries (**A**). After stenting, no pressure gradients were observed between the aorta and both the brachial arteries (**B** and **C**). Ao: aorta; Lt Br: left brachial artery; Rt Br: right brachial artery.

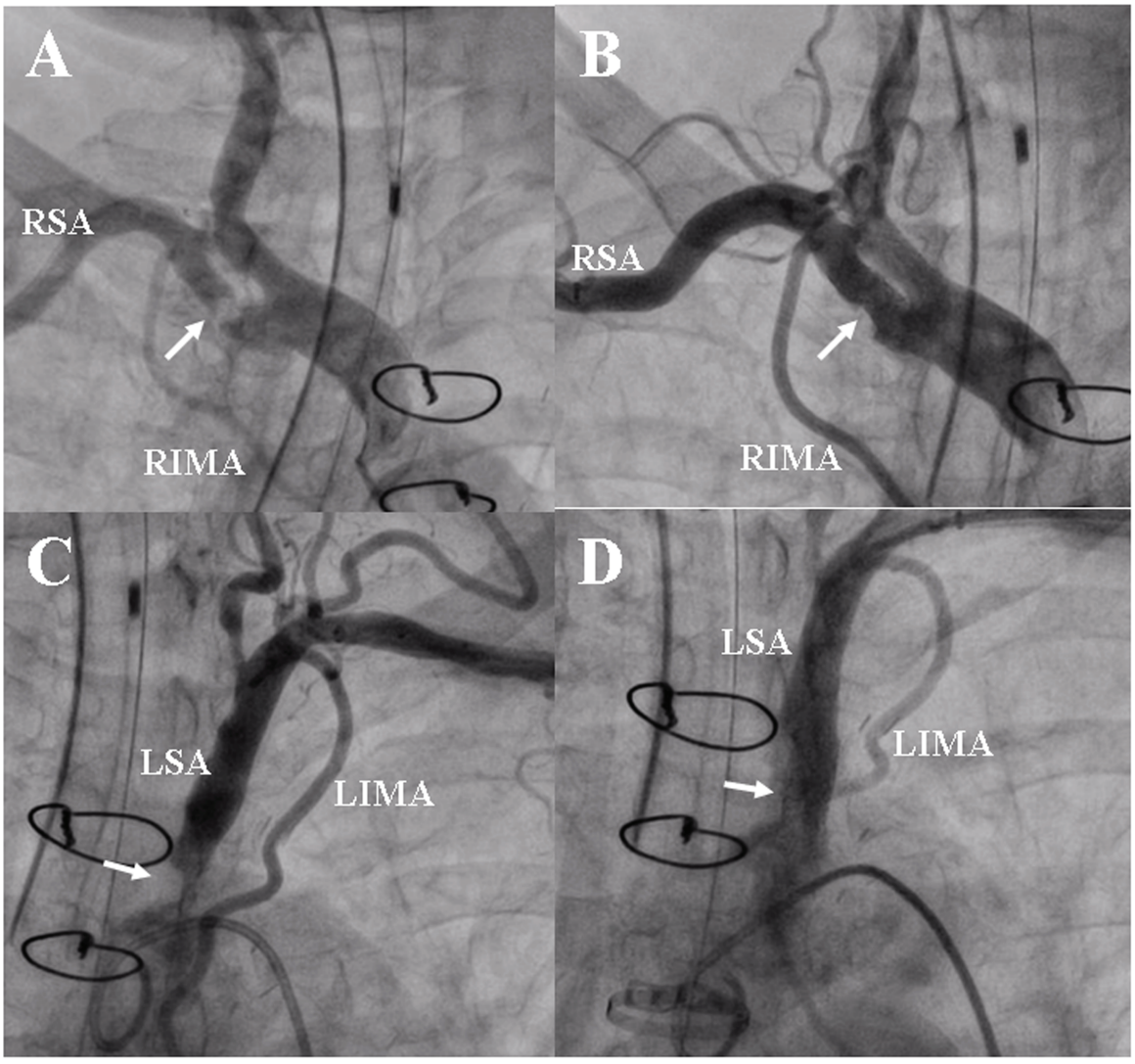

Figure 2: (**A**) Angiography revealed severe narrowing in the right subclavian artery (arrow) proximal to the RIMA that supplies to the obtuse marginal branch. (**B**) After stenting, the right subclavian artery is wide open with a minor residual indentation (arrow). (**C**) At baseline, there was a tight stenosis at the ostium of the left subclavian artery proximal to the LIMA that supplies blood to the LAD artery. (**D**) After stenting, there was no residual narrowing at the ostium of the left subclavian artery. Saphenous vein graft to the distal left circumflex artery was wide open (not shown). RSA: right subclavian artery; RIMA: right internal mammary artery; LSA: left subclavian artery; LIMA: left internal mammary artery.

stenosis in bilateral subclavian arteries located proximal to the IMAs (Fig. 2A and C). The native left main tract was 99% occluded, and the proximal LAD was completely occluded. RIMA and LIMA grafts were patent, and no coronary subclavian steal phenomenon was observed. Diffuse myocardial ischaemia due to low perfusion pressure in the bilateral mammary arteries was considered the cause of left ventricular dysfunction and pulmonary oedema.

TREATMENT

Following angiography, one 7-mm × 17-mm and another 6-mm × 14-mm Express SD stent (balloon expandable) was successfully deployed in the stenotic left and right subclavian arteries by retrograde approach (Fig. 2B and D). After the stenting, BP in both arms normalized, and no pressure gradient was observed (Fig. 1B and C). The patient's general condition improved, and she was extubated. She developed frequent episodes of ventricular tachycardia; hence, she was required to be on amiodarone and to have an implantable cardioverter-defibrillator, which successfully managed the refractory ventricular arrhythmia, possibly reperfusion arrhythmia. Gradually, her ventricular function improved, and she was discharged with no symptoms. Four years postoperatively, her clinical condition was shown to be stable. BP in both arms has remained uniform at about 120–130/70–75 mmHg, and there is no sign of congestive heart failure. The patient has not experienced any serious ventricular arrhythmias and enjoys regular exercise such as walking.

DISCUSSION

The incidence of SAS, defined as ≥10–15 mmHg inter-arm systolic BP difference, is 2% in the general population and can be as high as 8–18% or greater in groups at high risk such as those with peripheral artery disease [1–3]. Its most common aetiology is atherosclerosis, but it can also result from Takayasu arteritis,

compression syndrome and fibromuscular dysplasia [1–3]. The left subclavian artery is four times more likely to be affected than the right or innominate arteries [3]. Patients with SAS, as suspected by inter-arm BP difference, had a higher incidence of future cardiovascular events in a large community-based cohort [9]. Incidence of bilateral SAS, as in our case, is not well known and is considered very rare [1]. If bilateral SAS simultaneously progresses, as was the case in this study, it is difficult to identify SAS with an inter-arm BP difference [10]. The high ABI observed 1 year prior to admission, as in this study (represents the difference in BP of arms and legs), may be a key for accurately diagnosing bilateral SAS.

Symptoms related to SAS include arm claudication, pain while resting, finger necrosis from embolic debris and syncope due to subclavian steal syndrome [2, 3]. In a patient with CABG using IMA, angina pectoris may present due to decreased perfusion pressure [2, 3, 5, 6, 8]. Sometimes, myocardial ischaemia may arise from coronary subclavian steal syndrome (retrograde perfusion from IMA to the distal subclavian artery) [2, 3, 4, 7]. Various imaging tests including direct angiography, computed tomography angiography, MR angiography and duplex ultrasound with colour flow can be used for the diagnosis of SAS [1–7].

Remarkably, in this study, the bilateral SAS simultaneously progressed, leading to hypoperfusion of the bilateral IMA and global ischaemic myocardial dysfunction. Initially, the patient was considered to have cardiogenic shock; however, due to a discrepancy between BP and the clinical condition, a thorough physical examination was repeated. It revealed that the bilateral femoral arteries were palpable. Further, a review of the patient's history showed that she had moderate bilateral SAS as shown by MR angiography 2 years ago. In addition, her BP gradually decreased 2–3 months prior to admission. Upon considering these findings, pseudo-hypotension due to bilateral SAS [10] was suspected. Finally, angiographic imaging aided by intravascular ultrasound and pressure measurements across the stenoses exhibited haemodynamically significant blockage in bilateral subclavian arteries (Figs 1A and 2B and D).

In addition to a medical treatment with antiplatelets and statins, surgical treatment or EVT may be necessary for SAS before or after an IMA–coronary artery bypass surgery [2–8]. Angle *et al.* [8] reported 18 patients who required EVT for the left SAS that caused angina pectoris after LIMA–coronary artery bypass surgery. Three of these patients required EVT for left SAS before LIMA–coronary artery bypass surgery. Technical success was achieved in all patients, and the pressure gradient across stenoses improved from 29 to 3 mmHg after EVT. Surgical treatment includes using a free IMA graft, an RIMA graft or an aorto-axillary bypass [2–8]. At present, there have been no randomized controlled trials comparing EVT and surgery. In this study, balloon angioplasty followed by stent implantation was very effective. During balloon inflation, manual suction of the debris was performed from the long guiding sheath located just distal to the stent to prevent possible cerebral and peripheral embolism. After stenting, there was no pressure gradient, and the flow normalized (Figs 1C and D and 2B and C). Further, no embolic phenomenon occurred. To the best of our knowledge, EVT for concurrent bilateral SAS after bilateral IMA–coronary artery bypass surgery has never been reported.

In conclusion, patients undergoing IMA-CABG should be screened for possible SAS [2, 3, 4, 5]. Those with significant SAS should be treated by EVT before undergoing an IMA–coronary artery bypass surgery, and those with borderline stenosis should be periodically evaluated for possible progression of SAS following the surgery.

Learning Points

1. Simultaneous bilateral subclavian artery stenosis (SAS) resulting in diffuse myocardial ischaemic dysfunction post an internal mammary artery (IMA)–coronary artery bypass graft surgery (CABG) is rare but may present as pseudo-hypotension and congestive heart failure posing as cardiogenic shock.
2. Those patients undergoing IMA–CABG should be screened for possible SAS if they have bruit in subclavian area, inter-arm blood pressure (BP) difference or higher-than-normal ankle–brachial index (indicating BP difference between arms and legs).
3. Those who had significant SAS should be treated by endovascular treatment before undergoing IMA–coronary artery bypass surgery, and those with borderline stenosis should be periodically evaluated for possible progression of SAS following the surgery.

CONSENT

Written consent was obtained.

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