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CASE REPORT

An unusual cause of ischemia after coronary bypass grafting!!

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Abstract Coronary subclavian steal syndrome is an uncommon cause of ischemia recurrence after coronary artery bypass grafting. Endovascular treatment of subclavian artery stenosis or occlusion is increasingly common and appears to offer a safe and effective alternative to surgical revascularization. We report a case of recurrent angina after coronary artery bypass grafting for critical subclavian artery stenosis. The anomalous origin of the vertebral artery from the aortic arch was an indication for endovascular treatment. We discuss the diagnostic difficulties and the management pitfalls of subclavian artery angioplasty in this syndrome.

PALAVRAS-CHAVE
Angioplastia;
Síndrome do roubo da subclávia;
Enxerto da artéria coronária;
Caso clínico

Uma causa rara de isquemia após revascularização cirúrgica!!

Resumo A síndrome de roubo da subclávia é uma causa incomum de recidiva de isquemia após a cirurgia de revascularização do miocárdio (CABG). A terapêutica endovascular da estenose ou oclusão da artéria subclávia é cada vez mais usada e parece oferecer uma opção segura e efetiva à revascularização cirúrgica. Apresentamos o caso de uma angina recorrente após CABG devida a estenose arterial crítica da subclávia. A origem anómala da artéria vertebral a partir da crossa da aorta favoreceu o tratamento endovascular. Discutimos as dificuldades diagnósticas e as dificuldades do tratamento da angioplastia da artéria subclávia durante essa síndrome.

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Introduction

Angina recurrence in a patient after coronary artery bypass grafting (CABG) is usually attributed to graft dysfunction; coronary subclavian steal syndrome (CSSS) is rarely mentioned. CSSS is defined by retrograde blood flow from the left internal mammary artery (LIMA) into the subclavian artery (SCA); it is related to proximal stenosis or total occlusion of the SCA.[1] CSSS is not only difficult to diagnose but tricky to manage. Redux surgery is high risk in such patients and percutaneous treatment is sometimes made difficult by the angiographic appearance of the lesion. We report a case of CSSS and discuss diagnosis difficulties and management pitfalls.

Case report

Mr. M.K is a 55-year-old man with a medical history of hypertension, dyslipidemia, smoking, mild chronic renal disease and peripheral artery disease. In 2010, he had CABG of the left internal mammary artery (LIMA) to the left anterior descending (LAD) artery and a venous graft (VG) to the marginal. He remained asymptomatic for 6 years. Recently he developed chest pain on exertion, limiting his daily activities. He reported no other symptoms such as left arm claudication, paresthesia or dizziness. Physical examination was unremarkable, except for a difference in blood pressure between arms; left-arm pressure was 120/80 mmHg and right-arm pressure 140/80 mmHg. Left radial and brachial pulses were markedly reduced but we did not detect any abnormal murmur in the subclavian area. Electrocardiogram showed negative T waves in anterior leads. Echocardiography revealed a low left ventricular ejection fraction (LVEF=35%) with an anterior hypokinesia. Positron emission tomography (PET) showed anterior ischemia and inferior necrosis. A coronary angiogram was performed with initial right femoral access, showing a long severe stenosis in the LAD with competitive flow in the distal LAD and chronic total occlusions of the RCA and the first marginal. The saphenous-vein graft was also occluded. When trying to cannulate the LIMA, we failed to enter the subclavian artery. The aortogram revealed occlusion at the origin of the left SCA (Figure 1). We switched to left radial artery access; the LIMA was patent and free of stenosis. We decided to perform a transthoracic Doppler to check the flow in the LIMA, but the patient was lost to follow-up. Three months later, he returned with disabling angina. The transthoracic Doppler at that point showed retrograde flow in the LIMA, and confirmed CSSS. Computed tomographic angiography confirmed the presence of critical stenosis at the proximal SCA without any proximal stump (Figure 1); the aorta/clavicle junction was heavily calcified and the left internal carotid was very narrow with an ostial atheroma. Fortunately, the left vertebral artery originated directly from the descending aorta (Figure 2). After multidisciplinary team discussion and given the risk of redux surgery in a such patient, we opted for endovascular management. The procedure was not easy given the angiographic aspects of the risk of calcium embolism, the difficulty crossing the lesion and the proximity to the carotid artery. However, the vertebral artery originating from the descending aorta was a favorable factor for angioplasty.

We performed the angioplasty of the SCA via a retrograde access from the left radial artery. The crossing of the lesion was laborious; we used many wires and even coronary wires (Whisper MS, Pilot 150 and Miracle 4) (Figure 3). Finally, a TIF Tip™ 0.018 Terumo Hydrophilic Guidewire was advanced through the catheter and the SCA occlusion was barely crossed, using a retrograde subintimal dissection. Predilation using a coaxial balloon (Admiral Xtreme, Medtronic, 4×40 mm) was performed to the nominal diameter after confirming the intravascular position (Figure 3). Next, a balloon expandable stent (6×37 mm) was inserted without complications. A final proximal optimizing post-stenting angioplasty was performed. The final angiography showed a good result with TIMI III flow in the LIMA (Figure 3). Immediately after the procedure, the patient had a normal radial pulse. Six months later, his LVEF had improved (LVEF=45%).

Discussion

Nowadays, the LIMA graft is the most used as a result of the long-term patency and the low operative mortality rates.[2] CSSS is an uncommon complication and has a low reported incidence (0.2% to 6.8%) after CABG surgery with the LIMA[1,3,4]; the first case was described in 1966.[5] However, it seems to be underestimated and its incidence is currently rising as a result of the increasing use of the LIMA for CABG surgery.[1] CSSS is caused by stenosis or occlusion of the proximal SCA before the origin of the LIMA, resulting in reduced coronary flow and sometimes reversed flow. The consequence is myocardial ischemia.[1] SCA stenosis may be present before CABG surgery or develop subsequently as a result of atherosclerotic disease progression.[2] Thereby, the ischemic symptoms can develop immediately following the CABG surgery or up to 7-8 years later.[2] It is typically associated with signs of vertebrobasilar insufficiency.[1] Physical examination can reveal asymmetric upper-limb pulses and pressures as in our case or abnormal murmur in the subclavian area. The association of recurrence of ischemic signs and asymmetric systolic pressure after CABG should be suggestive of CSSS. This case highlights the importance of a systematic routine preoperative screening of SCA stenosis before CABG with either the LIMA or the right internal mammary artery, particularly in patients with multiple cardiovascular risk factors. SCA stenosis is typically diagnosed using continuous-wave Doppler ultrasonography on the LIMA; it will show reversed flow. Computed tomography, magnetic resonance imaging or angiography can be used as confirmatory tests for any suspected cases of subclavian steal. The traditional treatment consisted of surgical revascularization with extra-thoracic carotid-subclavian, subclavian-to-subclavian or axillo-axillary bypass grafting.[6] It was associated with excellent long-term patency and low mortality rates.[6] Currently, endovascular therapy including percutaneous transluminal subclavian artery angioplasty has emerged as a good alternative to surgery.[7,8] One of the largest studies including 170 patients who underwent stenting of subcla-