Case Report

A Combined Approach to Severe Multi-Organ Atherosclerosis

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Abstract

Severe coronary artery disease often coexists with peripheral vascular atherosclerosis. The assessment of the supra-aortic circulation is, therefore, of clinical relevance. We herein describe a case of coronary artery disease treated with surgical revascularization using the internal mammary artery and thereafter the progressive atherosclerotic disease of the native coronary arteries as well as the left subclavian and left renal arteries.

We also describe and discuss the clinical presentation, the diagnostic procedures, and the therapeutic approach with respect to the percutaneous transluminal angioplasty of the subclavian, renal, and right coronary arteries.

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Keywords: *Atherosclerosis • Subclavian steal syndrome • Coronary artery disease*

Introduction

Patients with atherosclerosis often have a diffuse manifestation of the vascular disease. Particularly, coronary artery disease is associated with a flow-limiting stenosis of the supra-aortic vessels.[1-3] Thus, in patients with previous coronary revascularization, either surgical or percutaneous and recurrent angina, the evaluation must include the native coronary circulation and implanted grafts together with the peripheral non-coronary circulation.[1]

This report describes a case of supra-aortic vessel disease with coronary-subclavian steal syndrome and the concomitant ostial lesion of the right coronary artery and left renal artery.

Case Study

A 65-year-old woman presented with a six-month history of progressive exertional angina and left upper extremity claudication, despite maximum medical treatment. She also reported left upper extremity pain, associated with dizziness and exacerbation of angina. She had no syncope. Her medical history was indicative of dyslipidemia and atherosclerotic heart disease with coronary artery bypass graft surgery 13 years previously. Her father died of myocardial infarction in his 70s.

On admission, the patient was on ASA, Plavix, Atenolol, Isosorbide dinitrate, Diltiazem, Atorvastatin, Gemfibrosil, and Captopril.

On physical examination, the blood pressure in the right and left arms was 170/90 and 80/55 mmHg, respectively. Additionally, the patient's PR was 80/min and her RR was 14/min, and she had an oral temperature of 37.2˚c. No carotid bruit and no other positive findings were detected in the head and neck. The patient's chest and lungs were clear to auscultation. A cardiac examination revealed prominent S4 and a systolic murmur with II/VI intensity at the left

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lower sternal border; the findings were otherwise normal.

The examination of the abdomen proved non-contributory. Weak and delayed pulse in the left radial, brachial, and axillary arteries was detected in the examination of the extremities. The findings of the neurological examination were within the normal limits.

The patient's laboratory findings on admission were as follows: glucose=100 mg/dl, K=3.8, creatinine=1 mg/dl, blood urea nitrogen=19 mg/dl, hemoglobin=13 g/dl and platelet count=260000×109 /L.

ECG demonstrated ST-T abnormalities in the precordial leads (V2→V4). The cardiothoracic ratio was in the upper limit of normal on chest X-ray. Transthoracic echocardiography revealed preserved left ventricular systolic function (ejection fraction=50%) with diastolic dysfunction (impaired relaxation pattern) and septal wall hypokinesia; moderate tricuspid regurgitation; mild mitral regurgitation; moderate aortic insufficiency; and mild pulmonary insufficiency. The estimated pulmonary artery pressure was 40 mmHg.

A Computed Tomography angiography was performed, which showed that the left anterior descending had some narrowing at the proximal part with cut-off at the mid portion. In addition, the left circumflex coronary artery had 90% stenosis proximally, and the right coronary artery had significant stenosis at the origin and another 50% stenosis before the patent ductus arteriosus branch. The saphenous vein graft on the obtus marginatum was patent.

The patient also underwent cardiac catheterization, which revealed a normal left main trunk, narrowing at the proximal part with cut-off at the mid portion of the left anterior descending, 90% stenosis at the proximal part of the left circumflex coronary artery, 95% calcified ostial stenosis of the right coronary artery, 50% stenosis before the patent ductus arteriosus branch, and patent saphenous vein graft on the obtus marginatum.

An angiogram of the aortic arch and great vessels (Figure 1) demonstrated Type 1 arch, normal right internal carotid artery, normal left internal carotid artery, and occluded left subclavian artery. The patient's left renal artery angiogram was indicative of severe left renal artery stenosis.

The most important step in the management of a patient is the identification of the cause of symptoms. Therapeutic options at this point in the presence of failing medical therapy include repeat surgery or percutaneous transluminal angioplasty of the left subclavian artery. The decision was made to proceed with angioplasty and stenting of the left subclavian.

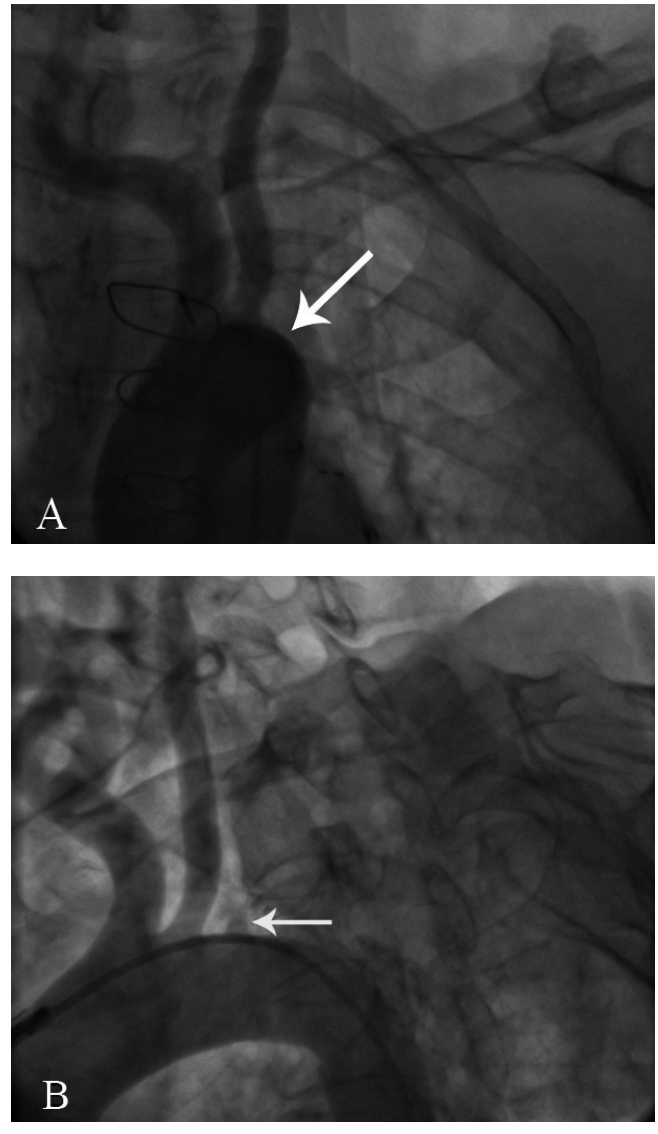


Figure 1. Angiogram of the aortic arch and great vessels in the anteroposterior (A) and lateral (B) views showing total occlusion of the left proximal subclavian artery (arrows)

Procedures

Procedure 1: Left subclavian revascularization

An approach was undertaken from the right transfemoral artery. Unfractionated heparin was administered to achieve an activated clotting time>250 seconds. A 6-Fr MP guide (Cordis, Miami, U.S.A.) was used to engage the ostium of the subclavian artery. A 0.014 BMW wire (Guidant, Santa Clara, U.S.A.) was employed to cross the lesion.

Angioplasty was performed with a 3.75×12 mm Maverick Balloon (Boston Scientific, M.A., U.S.A.) at 12 atm (Figure 2). The post-balloon angioplasty angiogram is shown in Figure 3. A 5×18 mm Genesis Stent (Cordis, Miami, U.S.A.) was subsequently deployed at 10 atm successfully with no residual stenosis, no dissection, and normal flow (Figures 4 and 5). Immediate equalization of blood pressure in both arms was acquired. Left radial approach was used for an evaluation of the left internal mammary artery, which showed a normal antegrade flow of the artery (Figure 6).

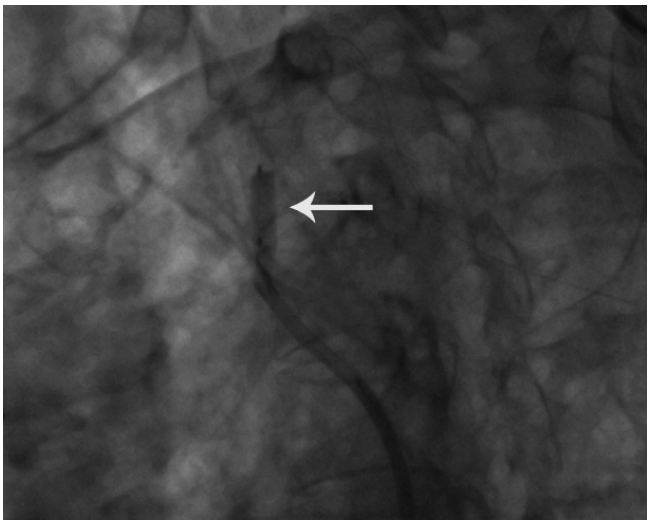


Figure 2. Angiogram showing a 3.75×12 mm Maverick Balloon (Boston Scientific, MA, U.S.A.) in the proximal part of the subclavian artery (arrow)

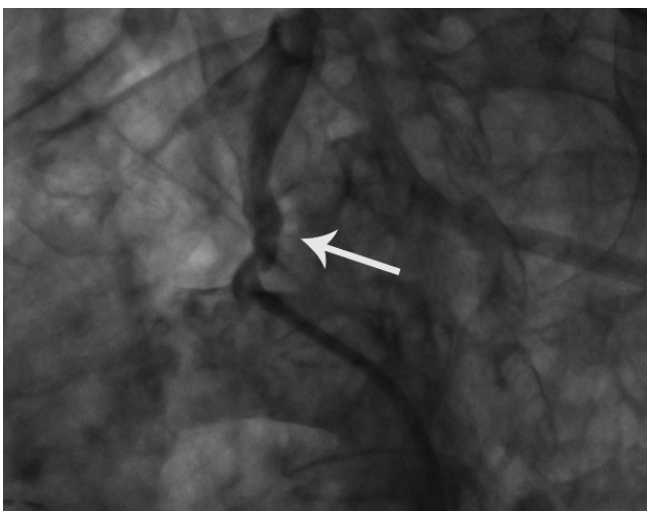


Figure 3. Angiogram showing the resolution of the proximal subclavian artery stenosis after the balloon angioplasty (arrow)

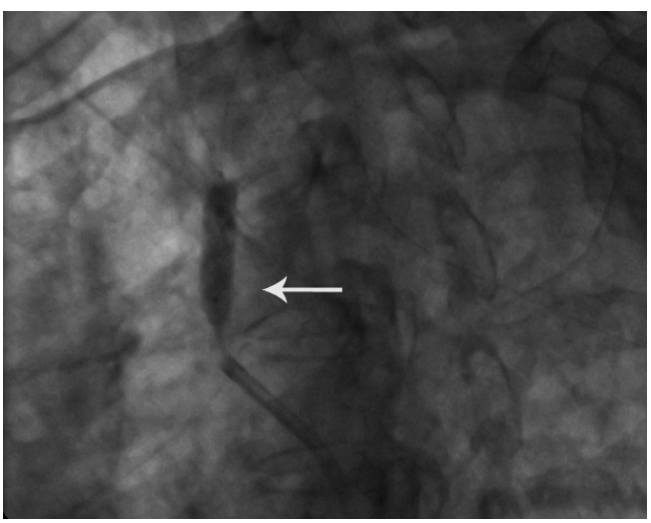


Figure 4. Angiogram showing a 5×18 mm Genesis Stent (Cordis, Miami, U.S.A.) in the proximal part of the subclavian artery (arrow)

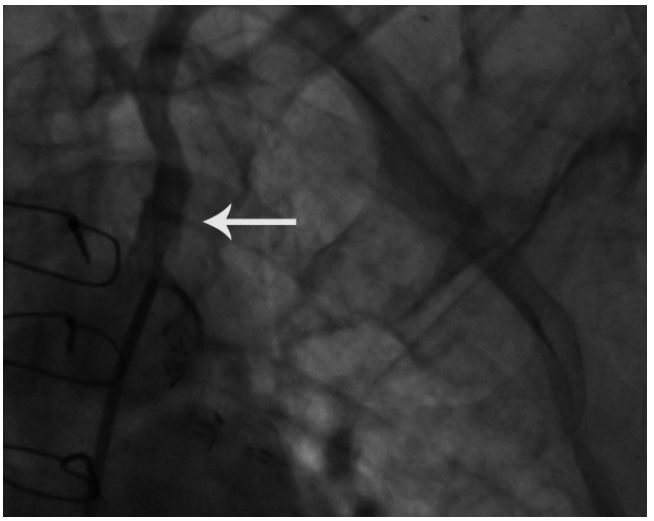


Figure 5. Angiogram of the stent being deployed within the left proximal subclavian artery (arrow)

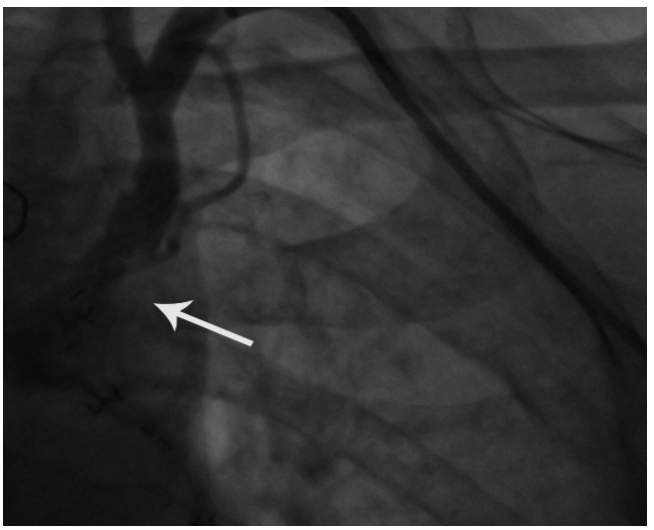


Figure 6. Left radial approach was used for evaluation of the left internal mammary artery, which showed a normal antegrade flow of the artery (arrow)

Procedure 2: Left renal artery revascularization (5 days later)

A 6-Fr renal double-curve guide (Cordis, Miami,U.S.A.) was used to employ a BMW guidewire (Guidant, Santa Clara, U.S.A.) in order to cross the left renal artery stenosis (Figures 7 and 8). The lesion was predilated with a 3.75×15mm Maverick Balloon (Boston Scientific, M.A., U.S.A.) at 6 atm (Figure 9). A 6×15 mm Genesis Stent (Cordis, Miami, U.S.A.) was deployed at 10 atm (Figure 10). A final angiogram revealed no left renal artery residual stenosis as well as a normal flow (Figure 11). Despite the debatable benefits of unilateral renal intervention, we opted to treat it given the patient's documented uncontrolled hypertension despite being on multiple antihypertensive medications.

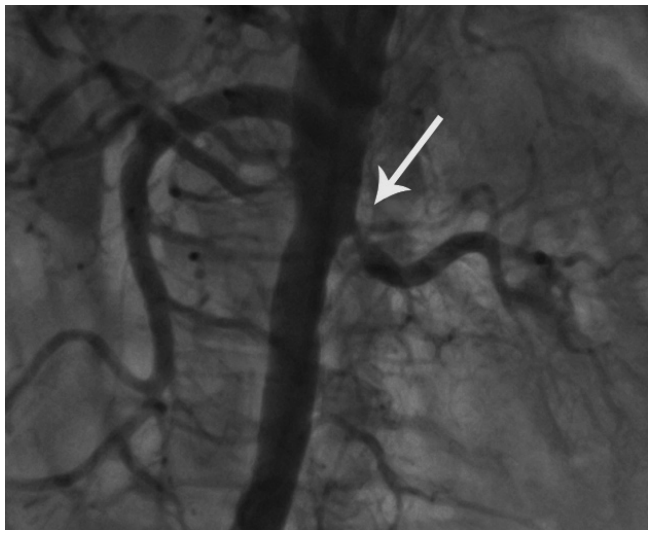


Figure 7. Angiogram of the abdominal aorta showing severe left ostial renal artery stenosis (arrow)

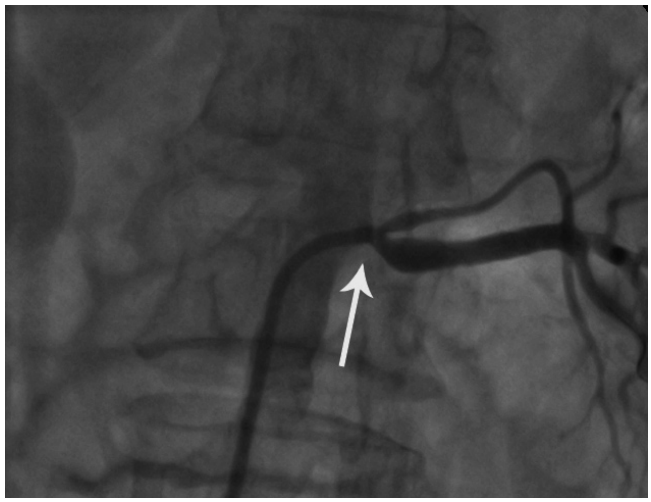


Figure 8. Angiogram showing the selective engagement of left renal artery using a 6-Fr renal double-curve guiding catheter (Cordis, Miami, U.S.A.) (arrow)

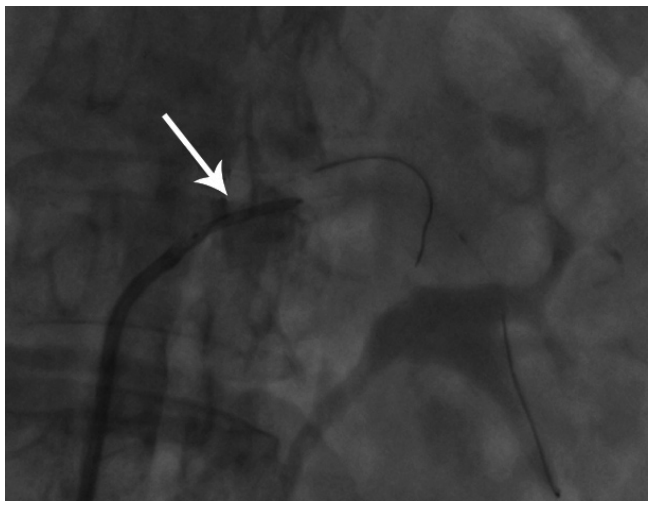


Figure 9. Angiogram showing a 3.75×15 mm Maverick balloon (Boston Scientific, MA, USA) in the ostial part of the left renal artery (arrow)

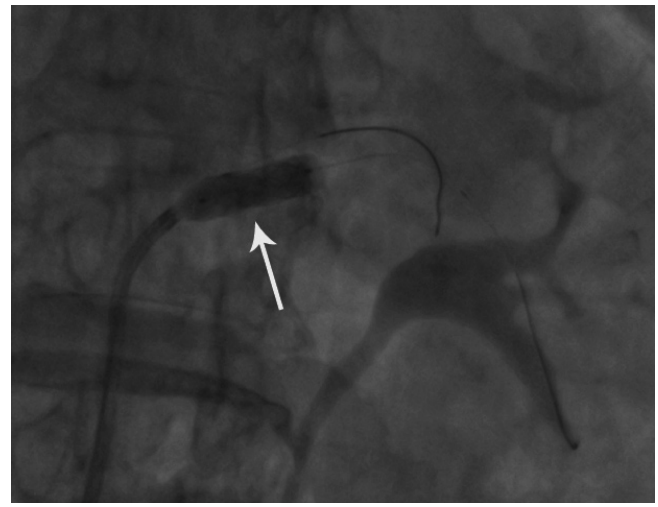


Figure 10. Angiogram showing a 6×15 mm Genesis Stent (Cordis, Miami, U.S.A.) in the ostial part of the left renal artery (arrow)

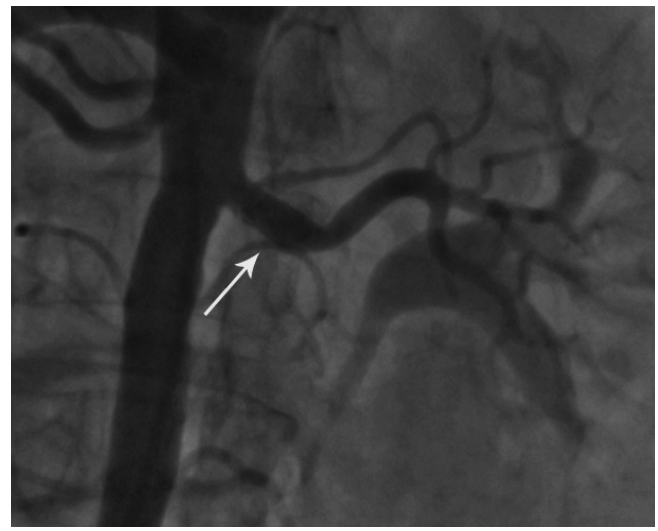


Figure 11. Angiogram of the stent being deployed within the left renal artery (arrow)

Procedure 3: Right coronary artery revascularization

The right coronary artery was engaged using a 6-Fr JR4 guide (Cordis, Miami, U.S.A.). Pressure damping was noted as soon as it was sitting on the right coronary artery ostium (Figure 12). A BMW guidewire was advanced into the distal right coronary artery. The guiding catheter was withdrawn slightly and kept in close proximity to the right coronary artery ostium along with the guidewire. Predilation was performed with a 2.75×9 Maverick Balloon and a 3.5×10 cutting balloon (Figure 13). Finally, a 3.5×13 mm Cypher Stent (Cordis, Miami, U.S.A.) was successfully deployed (Figure 14). A 3.5×10 Quantum Balloon (Boston Scientific, M.A., U.S.A.) was utilized to postdilate the lesion (Figure 15). The patient has done well since the procedure with better control of her blood pressure and no neurological or cardiac complications.

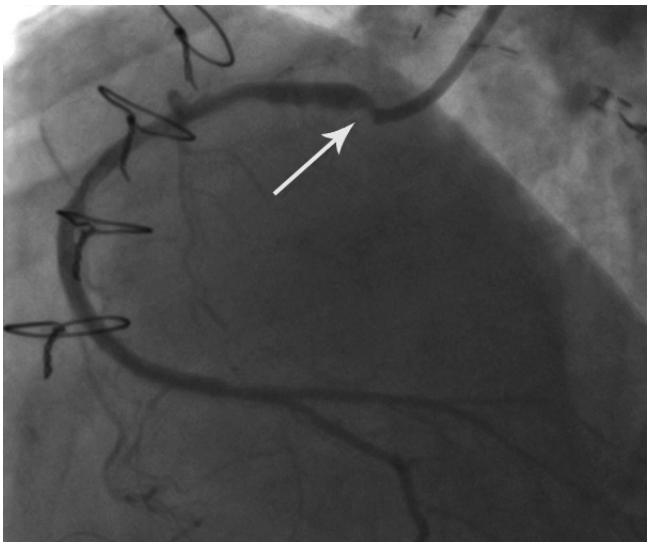


Figure12. Angiogram showing severe ostial stenosis of the right coronary artery (arrow)

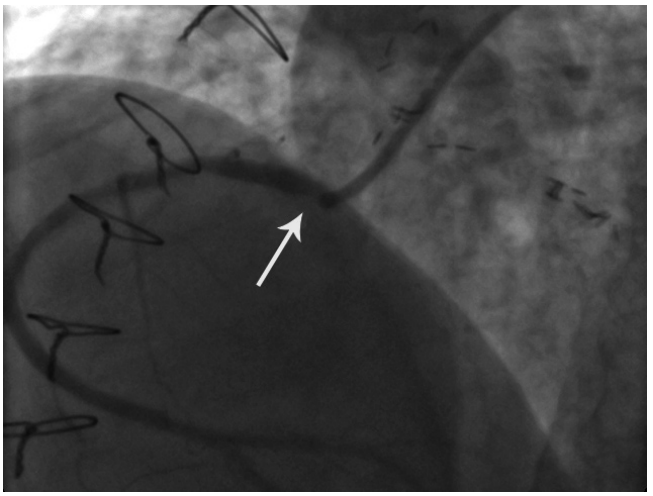
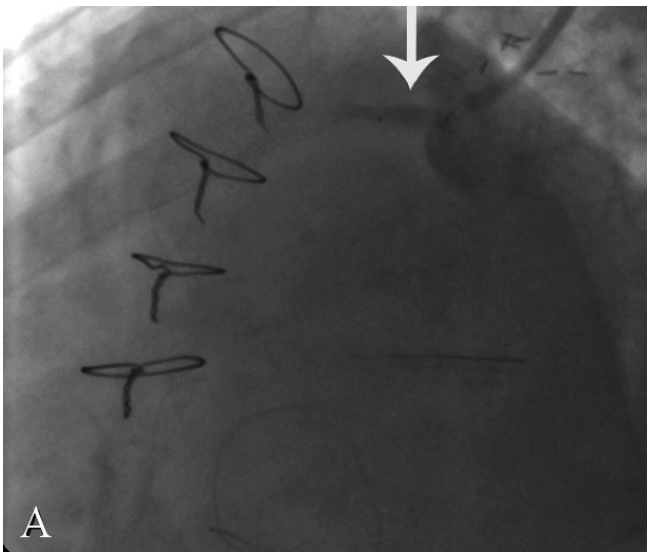


Figure 14. Angiogram showing a 3.5×13 mm Cypher Stent (Cordis, Miami, U.S.A.) in the ostial part of the right coronary artery and better resolution of the stenosis after the stent angioplasty (arrows)



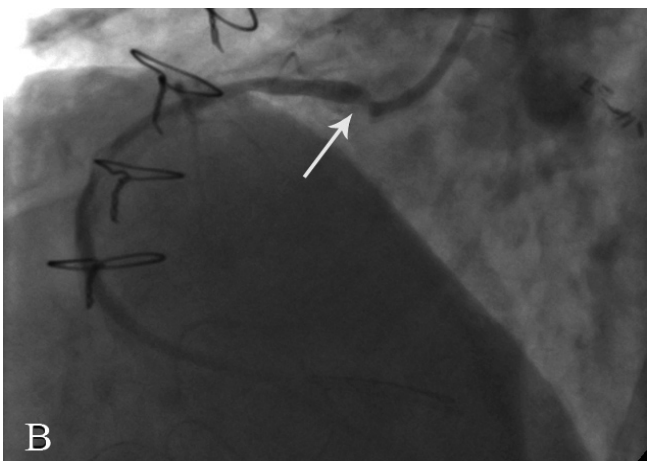
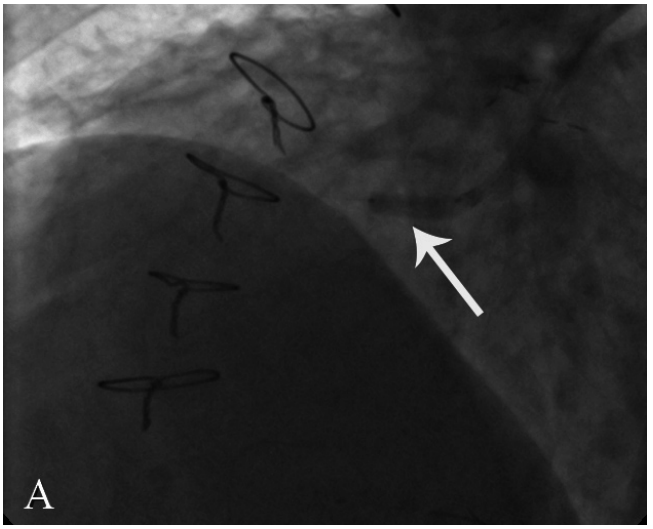


Figure 13. Angiogram showing a 3.5×10 mm cutting balloon (Boston Scientific, M.A., U.S.A) in the ostial part of the right coronary artery (A) and resolution of the stenosis after the balloon angioplasty (B) (arrows)



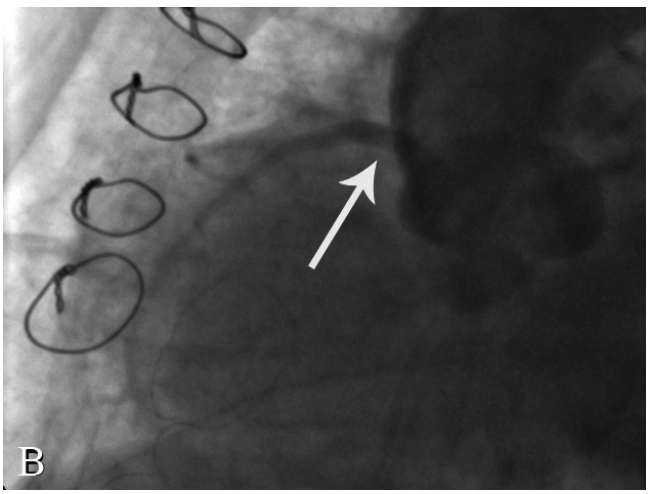


Figure 15. Angiogram showing a 3.5×10 mm Quantum Balloon (Boston Scientific, M.A., U.S.A.) used for postdilatation (A) and final result of the right coronary artery angioplasty

Follow- up

A duplex scan confirmed that there was no subclavian restenosis after 3 months' follow-up. The patient was asymptomatic, and she experienced no more angina as well as no more dizziness while working with her left arm. Systolic blood pressure at both arms was120 mmHg at rest.

Discussion

In daily clinical practice, the measurement of systolic blood pressure in both arms is recommended for hypertension screening.[4,5] This is to avoid a misdiagnosis in the case of lower systolic blood pressure in one arm, which may typically occur in case of subclavian stenosis, mostly because of atherosclerotic lesions occurring proximally, including the lesions of the innominate artery on the right side.[6]

Recently, two epidemiological studies[7,8] reported a significant correlation between subclavian stenosis and major cardiovascular disease risk factors such as age, smoking, and dyslipidemia. The presence of subclavian stenosis predicts total and cardiovascular disease mortality independent of both cardiovascular disease risk factors and existent cardiovascular disease at baseline.[9]

Coronary subclavian steal syndrome was first described by Hargola[1] and Tyras[2] in the 1970s. This syndrome is caused by atherosclerosis in most cases; nonetheless, it can occur in patients with a malformation of the left internal mammary artery, in the presence of large fistulas, in case of collateral vessels not adequately closed, arteritis, exterior compression, or iatrogenic arterial injury.[2,10]

The stenosis of the subclavian artery, proximal to the take-off of the left internal mammary artery, produces the inversion of the flow in left internal mammary artery itself and a steal of blood from the coronary circulation when this conduit is used as a bypass graft. The prevalence of significant subclavian arteries stenosis is low; it has been reported to be 0.4%.[2] In a recently published series, out of 780 patients treated with surgical myocardial revascularization, a concomitant occlusive disease of the subclavian artery was observed in 13 patients (1.6%).[11] This relatively uncommon cause of myocardial ischemia is, however, increasingly reported secondary to the more frequent use of the internal mammary arteries in cardiac revascularization.[12-14]

The typical manifestation of the syndrome consists of the recurrence of ischemia or angina despite a complete surgical myocardial revascularization, but it may also include other arterial territories, namely the carotid and vertebral-basilar. The stenosis of the subclavian artery also causes hypoperfusion to the ipsilateral arm, with dullness, pain, functional impairment, reduction in radial pulse amplitude, and decrease in blood pressure.[10-12] The reversal of the flow in the mammary artery can be enhanced by the vasodilatation in the ipsilateral arm during physical activity, but can occur at rest in case of tight stenosis.

The onset of symptoms in the first two years after surgery suggests the presence of severe stenosis at the time of surgery;[12] this underlines the importance of a complete pre-operative assessment of peripheral arterial circulation in patients with ischemic heart disease that must include the proximal portion of the supra-aortic trunks. A simple measurement of arterial blood pressure in both arms and finding of a difference may arouse the suspicion and guide the indication to further non-invasive or invasive testing.[15] A recurrence of symptoms indicative of subclavian artery stenosis more than two years after surgery is consistent with an obstruction originated or worsened after the pre-operative evaluation.

Conventionally, the treatment of coronary subclavian steal syndrome has been surgical via the creation of a carotid to subclavian or axillary-axillary bypass grafting or by the use of the internal mammary artery as a free graft on the ascending aorta.[16] Complications of the surgical treatment of subclavian stenosis have been reported in 5-20% of cases, including mortality in 5% of patients. The 5-year graft patency rate is 58-78%, and the most frequent non-fatal complications described are pleural effusion, cervical lymphatic fistula, wound infection, Horner syndrome, graft thrombosis, and stroke.[16]

Balloon angioplasty for subclavian artery stenosis was described in the early 1980s, with acute success and patency rates comparable to surgery. Successful revascularization was achieved in more than 90%.[17-19] Peri-procedural complications and strokes were uncommon. Primary patency ranged from 94% at 20 months to 75% at 8 years.[17]

As to the percutaneous treatment of subclavian stenosis, the immediate success rate approaches 100% of cases after stent implantation.[20] Complications associated with endovascular treatment occur in 5-14% of cases and are generally minor compared with those of surgical treatment; these include: hematoma of the puncture site, thrombosis, arteritis, and pseudoaneurysm. Both intimal dissection of the vessel at the dilation site and embolization through the vertebral artery with neurologic deficit are generally transient.[20,21]

The long–term clinical outcome of subclavian artery angioplasty seems to be independent of traditional risk factors for atherosclerosis. In a series of 91 consecutive patients evaluated retrospectively over a 9-year period, overall survival in the first 5 years was 93%, 88%, 84%, 81%, and 76%, respectively, and the overall clinical patency was 96%, 91%, 86%, 77%, and 72%.[20] Similarly, in a smaller series, clinical recurrence was observed in 15-30% of the patients over 2-year follow-up.[13,15]

In our case, we treated the patient by percutaneous balloon angioplasty and stent implantation of the subclavian artery seeing that the myocardial ischemia was due to the subclavian stenosis as a possible cause of blood flow steal. This approach is currently a valid alternative to surgical treatment achieving an optimal and immediate result while reducing the invasiveness, cost, and length of hospital stay.

Conclusion

The flow-limiting stenosis of the subclavian artery can be the cause of myocardial ischemia in patients with patent left internal mammary artery to coronary circulation. The percutaneous diagnostic and interventional approach is a safe and effective modality to identify and treat this infrequent pathology that is becoming an entity with increasing significance as the survival of patients with coronary artery disease improves.

The percutaneous coronary intervention of the aorto-ostial right coronary artery is difficult, not least when attempts are made to obtain a good guiding catheter support. It requires well-planned, quick technique of ballooning, and the procedure must be managed very swiftly. We herein presented a combined approach to severe multi-organ atherosclerosis.

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