

Case Report

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**An unusual culprit lesion in acute coronary syndrome:
In stent-restenosis of a subclavian artery stent****Boutaleb Amine Mamoun^{1,5*}; Scalia Alessandro²; Charaf Yassine³; Didier Wéry³; Carlier Stéphane^{2,3}**¹CHU Ambroise Paré, Mons, Belgium.²University of Mons, Belgium.³CHU Ambroise Paré, Belgium.⁴CHU Ibn Rochd, Casablanca, Morocco.***Corresponding Author: Mamoun BM**

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Abstract

A 73-year old man with past medical history of coronary artery bypass grafting in 2012 and subclavian artery stenting in 2021 was scheduled for an ophthalmologic minor surgery requiring conscious sedation. During the surgery, he developed hemodynamic instability and shortness of breath requiring the conversion to general anesthesia with mechanical ventilation. An electrocardiogram was immediately realized revealing deep negative T waves in all the precordial leads. Elevated troponin level confirmed the diagnosis of non-ST elevation myocardial infarction. Urgent coronary artery angiography revealed a known left coronary system with a chronic Left Anterior Descending (LAD) artery occlusion, a chronic occlusion of the distal circumflex artery and a severe stenosis of left main stem. The right coronary artery presented a significant and calcified stenosis on its mid part. The two venous grafts on the obtuse marginal and the posterolateral artery were both patent. The left subclavian artery could not be crossed to selectively film the left internal mammary artery and an angiogram revealed a tight stenosis in the previous stented segment, limiting the blood flow in the left internal mammary artery. Careful review of the in-stent restenosis suggested neointimal hyperplasia and stent fracture, as detected by stentboost imaging.

Keywords: Coronary subclavian steal syndrome; In-stent restenosis; Stent fracture; Myocardial infarction; Neointimal hyperplasia.

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Introduction

Myocardial infarction is defined as myocardial cell death due to prolonged ischemia. The European Society of Cardiology has distinguished four types of myocardial infarction with a particular pathophysiological mechanism for each one.

Following latest guidelines, Myocardial Infarction (MI) is defined as a rise of cardiac troponin markers associated with either symptoms of acute myocardial ischemia, new ischemic ECG changes, new loss of viable myocardium or new regional wall motion abnormality [1]. Coronary artery revascularization should follow the latest European Society of Cardiology guidelines. For patients with diabetes with multivessel disease, two vessel disease including proximal left anterior descending coronary artery bypass graft is recommended [2]. Coronary Artery Bypass Grafting Surgery (CABG) is an effective method to treat coronary artery disease. The revascularization technique is based on bypassing coronary artery obstructions using either the Internal Mammary Artery (IMA), a reversed Saphenous Vein Graft (SVG) or a radial graft. The majority of the patients undergoing CABG receive a Left Internal Mammary Artery (LIMA) to the Left Anterior Descending Coronary Artery (LAD) because of the long-lasting graft patency and survival benefits. In the context of a ST Segment Elevation Myocardial Infarction (STEMI), urgent percutaneous revascularization is required [3]. An acute coronary syndrome might be triggered by the disruption of an atherosclerotic plaque, plaque erosion, calcium nodules, vasospasm or coronary embolism. Without complete coronary obstruction, there is a Non-ST Segment Elevation Myocardial Infarction (NSTEMI) that requires more or less urgent coronary angiogram in function of clinical status [4]. The potential complication after any revascularization technique is anginal recurrence. It can either be related to progressive stenosis in the graft or the native coronary artery system.

Coronary subclavian steal syndrome is a rare complication of CABG when a LIMA graft is used. This syndrome is characterized by a retrograde flow from the LIMA to the Left Subclavian Artery (LSCA) when a proximal stenosis of the SA is present. As a result, the myocardium originally perfused by the LIMA graft can become ischemic despite the patency of the grafted vessel.

Atherosclerotic disease of the proximal LSCA is the most common cause of such a coronary artery steal syndrome. However, several other pathologic processes, such as Takayasu arteritis and radiation arteritis can be found [5]. The screening pre and post- CABG should include clinical investigation and imaging to prevent it. [6].

We report here a unique case of coronary artery steal syndrome related to a subclavian artery in stent restenosis in the context of stent fracture.

Case presentation

We report the case of a seventy-three-year-old man who underwent coronary artery by-pass grafting (LIMA to LAD) and two venous grafts to the posterior left ventricular artery and obtuse marginal branch in 2012. The patient's past medical history includes treated arterial hypertension, lower limb atherosclerotic disease requiring stent implantation in his right superficial femoral artery in 2020 and sick sinus syndrome treated

with the implantation of a dual chamber pacemaker in 2020. In 2021, he underwent a coronary angiography for ongoing chest pain revealing an occluded proximal LAD and patent LIMA on mid LAD, severe stenosis of the distal left main, significant and calcified stenosis of the mid right coronary artery, patent venous graft on the obtuse marginal, and a patent venous graft on the distal right coronary artery. Supra aortic trunk angiography showed a post ostial left subclavian artery occlusion with blood flow inversion in the vertebral artery. Hence, a 8 x 27 mm stent was implanted at the site of SA occlusion with good blood flow normalization through the LIMA and left humeral artery.

The patient had been scheduled for an ophthalmologic surgery without a cardiological preop workout examinalional. Pre-anesthesia consultation found a stable patient with good hemodynamic parameters. No chest pain was reported during the examination. Preoperation ECG showed a sinus rhythm at 65 bpm with no abnormal repolarization signs. The last echocardiography realized three months before surgery revealed a preserved left ventricular systolic function with 55% left ventricular ejection fraction and a moderate aortic valve stenosis. Considering the low surgical risk and the good functional capacity of the patient, no further tests were performed before intervention, but no blood pressure assessment at each arm was checked.

Induction anesthesia drug used was Propofol and sufentanyl causing a rapid decrease in blood pressure to a minimum of 90/65 mmHg requiring the administration of two liters of crystalloids. The patient then developed shortness of breath rapidly evolving to an acute pulmonary edema treated with mechanical ventilation and administration of diuretics. Post anesthesia ECG showed an auricular stimulated rhythm and deep circumferential negative T waves (Figure 1). Biological tests revealed an elevated cardiac Troponin levels at 2209 ng/ml and acute renal failure with a decrease of the glomerular filtration rate at 39 ml/min/1,73 m².

The patient was diagnosed with very high risk NSTEMI because of the acute heart failure symptoms and the coronary angiogram was realized following the ESC guidelines in less than 2 hours.

Coronary artery angiography was realized immediately with right femoral access and a 6 French introducer. The angiogram revealed patent coronary artery grafts (Figures 2,3,4) and a significant in stent restenosis of the subclavian artery due to a stent fracture (Figures 5,6) and neointimal hyperplasia at this site.

In this acute setting, the stenosis was predilated with a balloon of 8 mm diameter and a 9 x 38 mm stent was implanted with a good angiographic result (Figure 7).

Immediately after stent implantation, there was an improvement in the hemodynamic parameters. Arterial pressure became 110/65 mmHg. The patient was extubated three days after intensive care measures including diuretics and oxygen therapy allowing resolution of the pulmonary edema.

Dual antiplatelet therapy with aspirin 80 mg per day and clopidogrel 75 mg was reintroduced for one month to prevent any thrombotic complication following the stent implantation.

After 48 h of intensive care unit monitoring, the patient was transferred to the cardiology ward for follow-up. The control echocardiography revealed a preserved left ventricular function as compared with the pre-operative echocardiography.

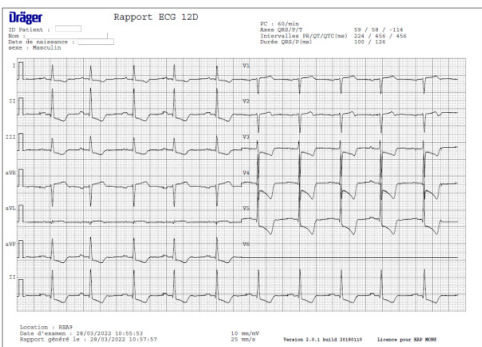


Figure 1: Post anesthesia ECG showing deep circumferential negative T wave and ST segment and decreased ST segment in septopicalateral leads.

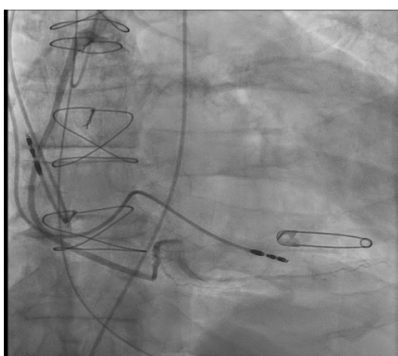


Figure 2: Coronary angiogram showing patent right venous graft on the distal right coronary artery.

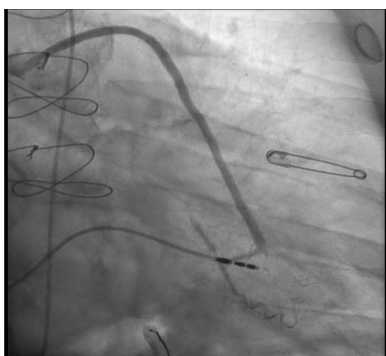


Figure 3: Patent saphenous vein graft on the obtuse marginal.

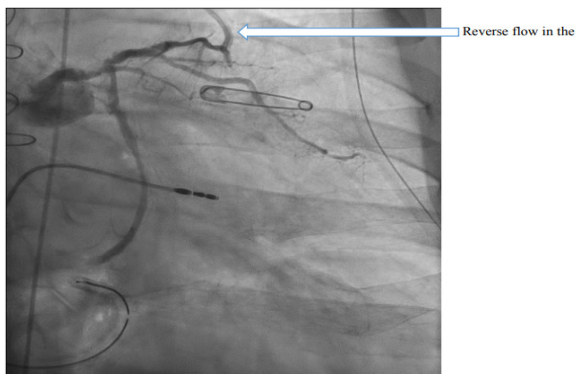


Figure 4: Coronary angiogram of the left coronary system showing significant left main coronary artery lesion and patent circumflex artery and LAD LIMA with reverse flow in the LIMA.

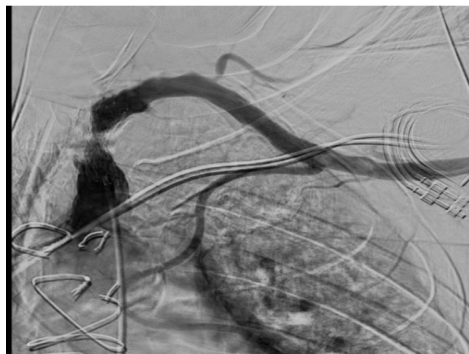


Figure 5: Severe in-stent restenosis of the left subclavian artery.

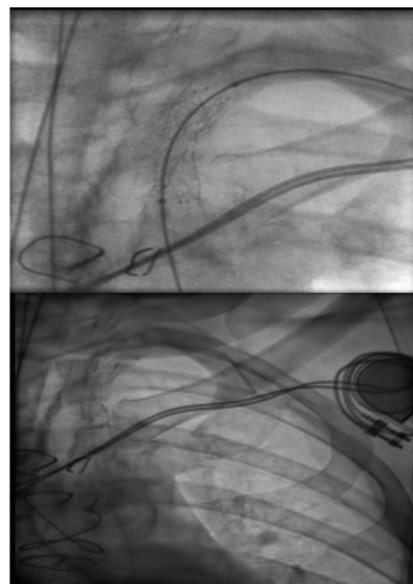


Figure 6: Fluoroscopy showing mid-stent fracture.



Figure 7: Left subclavian artery fluoroscopy control after stenting at the site of the stent fracture showing good result.

Discussion

This case report is a relevant cause of type 2 myocardial infarction caused by the disturbance of the oxygen balance between intake and consumption. This rare cause of AMI should be in mind in the context of CABG.

A threshold of 10 to 15 mmHg of differential arterial pressure between the two arms should lead to a computed tomography angiography and a supra aortic trunk arteriography to screen for subclavian artery stenosis. This threshold should be lower in the context of chest radiation therapy or vasculitis or peripheral artery disease [7].

A different arterial pressure between both arms is a known cardiovascular risk factor and indicator of risk of vascular disease and death [8]. It is recommended to measure the arterial

pressure at the two arms in patients after a CABG. If abnormal, echography and Doppler of the subclavian artery might show an accelerated flow and is the diagnostic noninvasive modality of choice. Turbulent flow and aliasing as reversal of ipsilateral vertebral flow are suggestive of subclavian artery stenosis. These echographic findings should lead to computed tomography angiography or angiography of the supra aortic trunks. Several methods of treatment of subclavian artery stenosis are possible. The endovascular approach with stenting is a less invasive procedure with good short and long outcome [9] and is preferred to the surgical procedure that can either be attempted via transposition of subclavian to carotid artery or subclavian bypass using either the carotid artery or the axillary artery [10].

In the patients, presenting with a medical past history of CABG, coronary- subclavian steal syndrome is defined by retrograde flow from the LIMA to the left subclavian artery occurring in the context of a proximal occlusion of the LIMA implantation site [11]. The retrograde blood flow to the left arm is responsible of myocardial ischemia symptoms.

The treatment of subclavian coronary steal syndrome immediately improves functional symptoms and clinical outcomes of these patients. A recent study reported that subclavian artery stenting of patients undergoing LIMA CABG is safe and carry low rates of complications and in-stent restenosis at 10 years follow-up [12].

ESC 2017 guidelines support endovascular revascularization of SA stenosis limiting to the surgical indications to failed endovascular procedures in low surgical risk patients [13]. Stent fracture can occur when compression and flexion forces applied to the stent prevail over its flexibility. Stent fracture classification varies between authors. Alqahtani & al reported multiple stent fracture classifications [14].

Our case report can be classified as a type III stent fracture upon the Jing Kan & al classification [15]. Stent fracture invariably leads to stent thrombosis and restenosis. The broken segment of the stent causes mechanical inflammation of the vessel wall leading to neointimal hyperplasia and either to in stent restenosis or stent thrombosis [16]. Coronary artery in stent restenosis is defined as a luminal loss after Percutaneous Intervention (PCI). The incidence of this complication has decreased with the last decade thanks to the newest generation of drug eluting stent. The major three pathogenic mechanisms are the elastic recoil, the vascular remodeling and neointimal hyperplasia [17]. Stentboost and intracoronary imagery with IVUS and OCT allow the physician to understand the underlying mechanism leading to the in-stent restenosis. Lesion revascularization is the cornerstone treatment for Stent Fracture (SF) with stent reimplantation for type III and IV SF and balloon angioplasty for lower risk SF types [15].

After reviewing the literature, we report the only case of subclavian artery stent fracture leading to coronary subclavian artery steal syndrome and NSTEMI. In a small series published by Miklós & al, the restenosis and reintervention rates were significantly higher in patients with type III-V fractures compared to those with type I-II fractures (60.0% versus 3.6%, $P < 0.001$; 46.7% versus 3.6%, $P < 0.001$, respectively) [18].

Cardiologists and anesthesiologist and should keep this rare complication in mind, in order to avoid more complications in this type of frail patients.

Conclusion

Acute myocardial infarction is often related to the occlusion of a native coronary artery. In the context of patients who underwent CABG receiving either a LIMA to LAD or RIMA to diagonal, it is important to look for supra aortic trunk stenosis. Clinical examination and radiological investigations are the cornerstone for early detection of this rare myocardial infarction cause. Stent fracture is a rare complication but an important factor of in stentrestenosis. Its treatment relies on either stent in stent implantation or balloon angioplasty depending on stent fracture classification. Target lesion revascularization improve outcomes of the patient presenting this complication.

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