Iatrogenic Aortic Dissection in Left Main (LM) Coronary Artery Percutaneous Coronary Intervention (PCI): Case Report
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Abstract

A 87 year-old hypertensive woman was admitted to our emergency department with NSTEMI diagnosis. Coronary angiography showed a 90% Left Main Coronary Artery (LMCA) calcified lesion and a 90% proximal LAD lesion. Angioplasty was planned in the same procedure, using radial approach 6Fr, we tried first a Launcher Extra Back Up (EBU) 3.5 6Fr guide catheter but for the lack of support, we changed for an Amplatz Left I (AL I) guide catheter. Successful reperfusion of the LAD lesion was achieved with a 2.5 mm x 33 mm Drug Eluting Stent, then we managed to treat the LM lesion ; Just after inflating a small ballon 3.0 x 12 mm in the ostium of the LMCA, we observed in angiographic control that contrast agent was stranded in the proximal area and spread outside of the aortic wall. Urgent aortic computed tomography angiography (CTA) showed that the dissection expanded to the ascending aorta and a large hematoma compressing the arterial pulmonary trunk. The patient was then transferred to the Department of Cardiac Surgery ; Decision for surgery was postponed and close monitoring leads to improved patient’s condition. Control CTA revealed no persistent pericardial effusion and hematoma, the patient was thus discharged.

Keywords: PCI; NSTEMI; Acute aortic dissection.

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INTRODUCTION

Iatrogenic acute aortic dissection is a life-threatening complication and remains one of the biggest fear of the interventional cardiologist, it can occurs during any percutaneous coronary intervention, thranscatherel valve replacement and thoracic endovascular aortic aneurysm repair. In interventional cardiology. This complication remains rare with a cumulative incidence of 0.062% during both diagnostic and therapeutic interventional procedures [1]. Treatment strategies are still controversial between surgery and conservative management. We present a case of a patient who developed an iatrogenic acute aortic dissection during a Left Main Coronary Artery (LMCA) percutaneous coronary intervention (PCI).

CASE REPORT

A 87 year-old hypertensive woman was admitted to our emergency department with acute chest pain. The electrocardiogram showed anterior extensive inverted T waves in lead V1 to V6, without ST segment elevation. Cardiac troponins were positive (80 ng/L) with a normal renal function (creatinine: 60 μmol/L). Transthoracic echocardiography showed wide hypokinesia in Left Ascending Artery (LAD) territory. We decided to manage the case as high risk Non ST-Elevation Myocardial Infarction (NSTEMI). Coronary angiography showed a 90% Left Main coronary artery (LMCA) calcified lesion and a 90% proximal LAD lesion (Figure 1).
Angioplasty was planned in the same procedure, using radial approach 6Fr, we tried first a Launcher Extra Back Up (EBU) 3.5 and 3.75 6Fr guide catheter but for the lack of support, we changed for an Amplatz Left I (AL I) guide catheter. Successful reperfusion of the LAD lesion was achieved with a 2.5 mm x 33 mm Drug Eluting Stent (DES), then we managed to treat the LM lesion; Just after inflating a small balloon 3.0 x 12 mm in the ostium of the LMCA, we observed in angiographic control that contrast agent was stranded in the proximal area and spread outside of the aortic wall.

The patient’s blood pressure dropped (70/40 mmHg) with tachycardia, and he required rapid fluid replacement. We suspected a LMCA and aortic dissection (Figure 2).

In parallel to initial conditioning, a 4 mm x 12 mm DES was quickly deployed in the ostial area to cover the tear of the dissection.

The patient’s condition has now stabilized, the blood pressure was 140/70mmHg, and the heart rate was 60 beats/minute.

Urgent aortic computed tomography angiography (CTA) showed that the dissection expanded to the ascending aorta; A hematoma surrounding the ascending aorta, compressing the pulmonary artery (Figure 3) and a moderate pericardial effusion. The patient was then transferred to the Department of Cardiac Surgery, Arnault Tzanck
Institute, France; Decision for surgery was postponed and close monitoring leads to improved patient’s condition. Control CTA revealed no persistent pericardial effusion and hematoma (Figure 3), the patient was thus discharged.

Fig 3: In the left side: CTA showing that the dissection expanded to the ascending aorta (red arrow) and wide hematoma compressing the pulmonary artery trunk (blue arrow). In the right side: Subsequent CTA after close monitoring, showing no dissection and no hematoma.

**DISCUSSION**

Iatrogenic acute aortic dissection in interventional cardiology remains a rare complication with a cumulative incidence of 0.062% [1]. This complication is more frequent during PCI than in coronary angiography (<0.01%). The incidence is more important in the acute setting of myocardial infarction (0.19%) than in elective coronary angiography or PCI (0.01%) [2].

Chest pain is the main symptom [3]. It can manifest in a knife-like pain, tearing, ripping; however it is the abruptness of symptom onset that remains specific [4]. In our case, the patient didn’t report any symptom, and it was the abrupt drop in blood pressure and tachycardia that predicted the onset of the acute dissection.

Diagnosis of suspected IAAD relies on imaging examination. The National Heart, Lung, and Blood Institute classifies coronary-induced dissections into six types (A–F) based upon their CA appearance [5]:

(A) Minor radiolucent areas with little or no persistence of contrast; (B) Parallel tracts or a doubled lumen with little or no persistence of contrast; (C) Contrast outside of the coronary lumen with persistence of contrast;

(D) Spiral dissection with filling defects; (E) Persistent filling defects in the lumen; and (F) Total occlusion without distal flow.

Our case was classified Type C, with a small difference: Contrast spreads in the ascending aorta; it suggests that the entry point was focal and precisely located between LMCA ostium and the ascending aorta.

Few studies have reported isolated dissection of the aorta without involvement of coronary arteries [6]. After stabilization, CTA is the gold standard in the diagnosis of coronary and aortic dissection.

A lot of factors has been described as contributing to IAAD mechanism; however the mechanical explanation for the complication is that, during the intervention there is a retrograde propagation of a coronary dissection resulting from mechanical trauma due to an aggressive guiding catheter, wire progression, or inflated balloon [7]. One of the most important factors that can increase the risk of dissection is the nature of the coronary artery lesion, as calcified lesions, which require more aggressive guiding catheter [8].

In our case, the LMCA lesion was located in the ostium, close to the aorta wall, and also was calcified, even if we managed the case using an aggressive guiding catheter (AL I), to provide a better support, we assume that there was a soft manipulation of the catheter and the mechanism that leads to dissection was identified as the balloon inflation.

We believe that are some principles to follow when facing an IAAD. In case of IAAD secondary to an aggressive manipulation of the guiding catheter, it is advised to change the guiding catheter to avoid accidently intubation the false lumen. Minimizing contrast injection to slow down the progression of the dissection. Prompt deploying a stent to cover the tear of the dissection.
In this present case, we carefully followed all these steps, the patient’s condition was stabilized quickly, which improved considerably her prognosis.

**CONCLUSION**

Iatrogenic acute aortic dissection caused by PCI remains a rare complication; however, it remains one of the biggest fears of every interventional cardiologist. Thus, managing this complication is primordial. We described in this case report some of the essentials principles that any operator should follow as soon as possible.

**REFERENCES**


