Surgical Management of Type A Aortic Dissection After Percutaneous Coronary Intervention in a Patient with Acute Myocardial Infarction Due to a Spontaneous Coronary Artery Dissection

Athanasia Makrygianni [1], Filippos - Paschalis Rorris[1], Lydia Kokotsaki [1], Konstantinos Velissarios [1], Mohammad Salmasi[2], Thanos Athanasiou[2], and John Kokotsakis[1]

[1]Evangelismos Athens General Hospital
[2]Imperial College London

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Abstract

Type A aortic dissection after percutaneous coronary intervention is a rare and life-threatening situation. The reported incidence ranges from 0,02% to 0,6% of all diagnostic and interventional percutaneous procedures. We describe a case of aortic dissection after percutaneous coronary intervention in a patient with acute myocardial infarction from spontaneous coronary artery dissection. The patient was initially treated with primary percutaneous coronary intervention of the affected left coronary artery branches and left main coronary artery. Conservative management for the aortic dissection proved inadequate due to increasing diameter of the false lumen and the patient was referred to our cardiovascular unit for definite surgical management.

INTRODUCTION

Spontaneous coronary artery dissection is a rare condition defined as an automatic separation of the coronary artery wall. It is not iatrogenic, not related to trauma or atherosclerotic disease. Women are mostly affected (>70%).(1) Two mechanisms describe SCAD: a "primary" rupture of coronary endothelium with resultant dissection of the coronary artery wall or the rupture of the "vasa vasorum" with formation of an intramural hematoma (IMH). According to angiographic findings, there are three types of SCAD. Type I: characterized by contrast dye staining of the arterial wall with multiple radiolucent lumens. Type II: more diffuse (length >20mm) and usually presents uniform narrowing of the coronary vessels. Type III: focal or tubular (<20 mm) stenosis that mimics atherosclerosis. (2)

The following case illustrates a case of type II SCAD, and the subsequent evolution into a type A aortic dissection. The details of surgical management required to salvage the patient are then described, including ascending and hemi-arch aortic replacement.

CASE PRESENTATION

A 59-year-old female smoker with a history of hyperlipidemia was admitted for ST-segment elevation myocardial infarction (STEMI) in a peripheral hospital. Transthoracic echocardiography (TTE) revealed hypokinesis of the lateral wall, however systolic function was preserved (left ventricular ejection fraction (LVEF) 55%).

She was immediately transferred to the catheterization laboratory, where coronary angiography revealed type II spontaneous coronary artery dissection (SCAD) of the left coronary system, initiating from the distal

part of the left main coronary artery (LMCA) (Figure 1A B,). Initially stenting of the ramus intermedius was performed. During stenting manipulations of the first diagonal and left anterior descending (LAD) artery, the dissection extended retrogradely into the LM and left coronary sinus (Figure2A). An additional stent was therefore inserted to the LMCA until its ostium. Specifically, a 3.5mm x 26mm stent followed by overdistension with a noncompliant balloon 4mm x 2mm was introduced into the LMCA, with partial protrusion of its foci into the aortic lumen. Because of the unimpeded flow in the circumflex coronary artery, the dissection was left unhandled. No apparent residual stenosis was seen after the procedure (Figure 2B).

The following day, transthoracic echocardiography (TTE) identified a notable pericardial effusion leading to prompt pericardiocentesis, which evacuated 540cc of bloody content. An urgent CT angiography revealed an aortic intramural hematoma <1cm in size extending longitudinally along the anterolateral side of the ascending aorta, from the aortic root up to the origin of the innominate artery (DeBakey type II). The aorta was 44-45mm in diameter.

Despite attempts to manage this conservatively with strict blood pressure control on the coronary care unit, a repeat CT scan 12 days later revealed expansion of the intramural aortic hematoma, and residual pericardial effusion (Figure 3). Repeat TTE revealed worsening LV systolic dysfunction (LVEF 40%) and hypokinesis of the lateral and anterior-apical cardiac walls, suggesting compromised flow in the left coronary system. The patient was escalated and transferred to the cardiothoracic centre for definitive management.

Surgical management

A median sternotomy was performed. Cardiopulmonary bypass was established by arterial cannulation of the right axillary artery through an 8mm interposition Dacron graft and venous cannulation with a standard two-stage venous cannula. The innominate and left common carotid arteries were freed up with soft tissue dissection and snared (Figure 4A). After cross clamping the ascending aorta, cold crystalloid (Custodiol) cardioplegia was administered retrogradely through the coronary sinus as well as antegradely though the right coronary artery ostium. The dissected ascending aorta was transected and inspected, to find an entry tear superiorly and laterally of the left main coronary ostium in close proximity. The LM stent was also visible, protruding into the aortic lumen (Figure 4B). The crucial decision was made to preserve the aortic root and seal the entry tear using a single pledged 5-0 polypropylene suture. After reaching 25°C, CPB was arrested and the aortic cross clamp removed for inspection of the aortic arch. The origin of the epiaortic vessels as well as the aortic arch were intact. Unilateral selective antegrade cerebral perfusion was achieved through right axillary artery cannulation by snaring the innominate, left common carotid arteries and clamping the origin of left subclavian artery. A straight tube 28mm Dacron graft was used for ascending aortic reconstruction with hemiarch replacement. During the procedure, continuous cerebral oximetry monitoring with a pre-installed INVOS (Medtronic) was used.

The patient's postoperative course was uncomplicated, and TTE showed no evidence of left ventricular systolic dysfunction. At 8-month follow-up, CTA demonstrates no residual aortic dissection and patent coronary stents (Figure 5).

DISCUSSION

Percutaneous coronary intervention (PCI) in SCAD patients is associated with high rates of complications. Iatrogenic coronary artery dissection (ICAD) is very infrequent, but a tremendous complication after coronary angiography in the general population, <0,2%.[3] Prakash et al. found the incidence to be 2% during diagnostic coronary angiography alone and up to 14,3% among patients with PCI on SCAD. [3] PCI in the presence of SCAD is technically more challenging. Maneuvering inside the coronary arteries can provoke propagation of the dissection ante- or retrogradely no infrequently, which in turn results in expansion of the area of myocardial ischemia. Forced and intense injection of the contrast medium must be avoided. [4,5]

Although rare, iatrogenic retrograde extension of the dissection into the ascending aorta (0,02 – 0,6% of diagnostic and interventional percutaneous procedures) is reported.[6] Dunning et al. have found a significantly increased rate in aortocoronary dissections in procedures performed for acute myocardial infarction (AMI)

(0,19%) compared with non-AMI cases (0,01%).[7,8] To our knowledge, this is the first reported case of type A aortic dissection after PCI for AMI due to SCAD. More frequently, iatrogenic type A aortic dissection involves the right coronary artery because it is more susceptible to trauma from a guide catheter than the LM. [7] In our case, the dissection extended from the LM to the aorta because of preexisting SCAD in the left coronary artery system.

Aortocoronary dissection is usually suspected at the time of coronary intervention. According to Dunning et al. criteria, the aortocoronary dissection is distinguished into three classes: Class I when the aortic dissection involves the ipsilateral cusp. Class II, when it includes cusp and extends up to the aorta for less than 40mm and Class III as for Class II but at length over 40mm. [8] There are no guidelines for treating aortic dissection caused by PCI.[9] The best treatment in Classes I and II seems to be the stenting of the coronary artery, including the respective ostium trying to seal the entry point of the dissection. Then consider conservative management with intensive clinical follow-up. Additional imaging controls are necessary only in unstable patients and if there is evidence of progression of the lesion. In contract, Class III mandates more aggressive surgical management. [7,8,10] In our case, the presence of abundant pericardial effusion the day after PCI comprised a high index of suspicion of progression of the known retrograde aortocoronary dissection. The CT scan revealed a Class III aortocoronary dissection: dissection with IMH of the ascending aorta extending from the left coronary sinus to the inferior limit of the innominate artery origin (Figure 3A). Besides the fact that the patient was stable and there was no evidence of active flow of contrast media in the false lumen, the delay of surgery just reconfirmed the necessity of emergent intervention in Class III aortocoronary dissection. Indeed, as shown on the revaluation with CT scan on day thirteen, there was an increase of the aortic hematoma despite the initial invariable CT image on day eight of hospitalization.

CONCLUSION

Iatrogenic aortocoronary dissection after PCI on AMI patients from SCAD is a rare complication. The pathology is characterized by the tendency of evolution and progression as in an acute aortic syndrome. Especially for more extended dissections of the aorta (over 40mm in length), the immediate surgical intervention seems to be the most appropriate treatment. In the case of primary stenting of the left main coronary artery, if possible, the more conservative surgical management techniques reduce the risk of major complications generated from the mobilization of the rigid stented artery.

Ethics Statement

This manuscript and all of its content meet the ethical guidelines and obtained an ethical waiver from the study institution. Written consent for submission and publication of this case report including images and associated text has been obtained from the patient.

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FIGURE LEGENDS

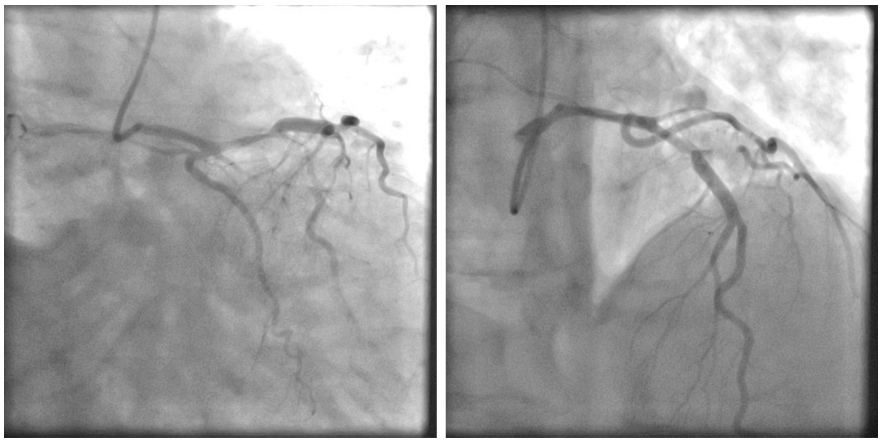
Figure 1: Different views of the spontaneous coronary artery dissection during PCI. Elongated uniform stenosis initiating from the distal part of the left main and extending mainly in the LAD and first diagonal as well as in the ramus intermedius.

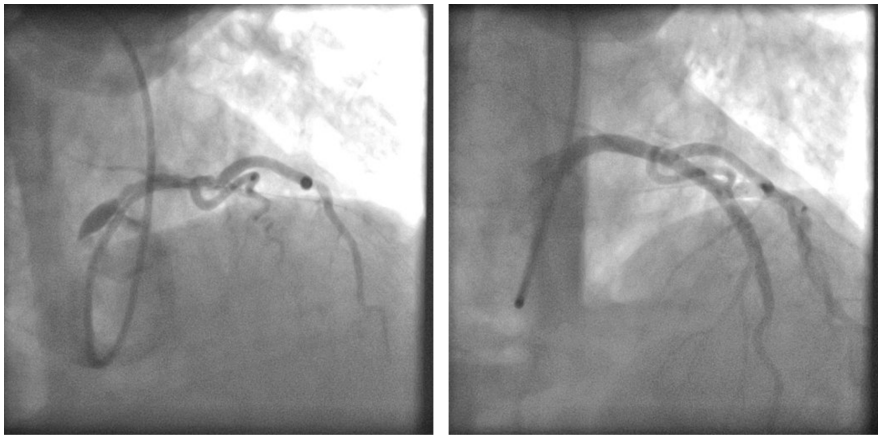
Figure 2: **(A)** Retrograde extension of the dissection into the proximal left main coronary artery until its origin in the aorta. A stent has already been positioned into the ramus intermedius.**(B)** Final angiogram after stenting of the LAD, D1 and LM to its origin, with no residual stenosis.

Figure 3: CT angiography imaging showing **(A)** the intramural hematoma (red arrow) just proximally to the origin of innominate artery and **(B)** the LM stent which protrudes into the aortic lumen.

Figure 4: Intraoperative images **(A)** Asc. Ao intramural haematoma, **(B)** LM stent protruding into the aortic lumen

Figure 5: 8-month f-up CTA demonstrating **(A)** no residual aortic dissection and **(B)** patent coronary stents.





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