Immediate Interventional Management of Aortocoronary Dissection During PCI: A Case Report

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Abstract

Aortocoronary dissection is a rare and potentially fatal complication of percutaneous coronary intervention. It is more common when performing interventions of the right coronary artery or during revascularization of chronic total occlusions. Here, we aimed to provide more data regarding a less common form of this complication. We present the case of an 81-year old woman admitted with an acute infero-lateral myocardial infarction, who was indicated for emergency primary coronary intervention. After revascularization of the left circumflex artery, the left main coronary artery exhibited retrograde dissection into the ascending aorta. This complication was managed with immediate placement of another coronary stent at the left main coronary artery. Further propagation of the dissection was prevented. The patient was discharged in good health with antiagregant and rhythm control therapy for new onset atrial fibrillation. Although retrograde dissection of the left main coronary artery is a very rare complication, it can be successfully managed interventionaly.

Keywords: Aortocoronary dissection; primary coronary intervention; myocardial infarction; case report

Introduction

Aortocoronary dissection is a rare and potentially fatal complication of percutaneous coronary intervention (PCI). The most important risk factors are age, hypertension, severe atherosclerosis and concomitant acute myocardial infarction (AMI) [1]. The dissection of a coronary artery can show either antegrade propagation with total lumen occlusion, or retrograde propagation toward the coronary cusps and the aorta [2]. Although its overall incidence is very low (<0.1%), this condition is more common in interventional than diagnostic procedures, and is associated with very high mortality [3]. Chronic total occlusion (CTO) revascularization procedures are more frequently associated with this type of complication [4]. The dissection of the left main coronary artery (LM) is relatively rare, and timely recognition and management are crucial. This report is aimed at providing more data on the topic, and summarizing the background and treatment strategies.

Case Description

We present the case of an 81-year old woman with chest pain onset 90 minutes before admission and subsequent transitory loss of consciousness. She had a history of hypertension and had experienced

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a single episode of colonic diverticular bleeding. Her medications included bisoprolol 5 mg bid and amlodipine 5 mg. On arrival, her vital signs were stable, but her ECG revealed a sinus rhythm with 3 mm ST segment elevations in leads II, III, aVF and V5-V6, and ST segment depressions in V1-V2. Her initial echocardiography evaluation showed preserved left ventricle size, kinetics and function (Figure 1). Although the initial troponin I dose was 0.2 mg/L, because of her clinical presentation and ECG signs for acute infero-lateral myocardial infarction, she was sent into the catheterization laboratory within 30 minutes after admission. She received 5000 IU of heparin (80 IU/kg; patient weight approximately 60 kg), 300 mg aspirin and 80 mg atorvastatin.

During catheterization, the left and right coronary arteries were evaluated with a 5 F Tiger diagnostic catheter (Terumo Corporation, Tokyo, Japan). The circulation was right dominant. The LM was intact but severely calcified. The left anterior descending artery showed one 50% stenosis in the middle segment and one 70% stenosis in the distal segment, where the diameter of the vessel was less than 2 mm. The left circumflex artery (LCx) was occluded from the ostium. The right coronary artery showed diffuse changes and stenoses reaching 70% in the middle segment.

The left coronary artery was engaged with a 6 French Extra Backup 3.5 Guiding Catheter (Cordis Corp., Miami Lakes, Florida). A Hi Torque Whisper LS Guidewire (Abbott Medical, Santa Clara, CA, USA), Sprinter 1.25/10 mm (Medtronic Inc, Minneapolis, MN, USA), Maverick 1.5/15 mm (Boston Scientific, Marlborough, MA, USA) and Accuforce 2.0/8 mm (Terumo Corporation, Tokyo, Japan) balloons were used to restore blood flow to the LCx. We proceeded with stenting the LCx with a DES Coroflex Isar Neo 2.25/12 mm (B. Braun, Melsungen, Germany). After expansion of the stent, the left coronary artery dissected at the LM, with the false lumen propagating upward into the left aortic sinus and the ascending aorta. The patient reported some chest pain but remained hemodynamically stable. An emergency decision was made to stent the LM with a DES Coroflex Isar Neo 4.0/12 mm (B. Braun, Melsungen, Germany), which prevented the further propagation of the dissection (Figure 2).

The emergency decision to stent the LM was made by the chief operator, on the basis of his previous experience, and was later supported by the other members of the heart team. The contraindications for surgical repair were the patient’s advanced age and a likely severely calcified aorta and LM. Our patient’s STS risk score indicated a 26.6% risk of operative mortality. A conservative approach might have resulted in greater complications necessitating subsequent, more extensive surgical repair of the aorta, which might have resulted in an even higher mortality risk.

The patient spent the next 5 days in the intensive cardiology unit. She was feeling well, without any chest pain. Her troponin I peaked at 20 mg/L, then steadily declined. Because 5000 IU heparin was also administered during catheterization, 1 hour after the initial bolus at admission, our strategy was to limit patient bleeding. She was loaded with a lowered dose of 300 mg of clopidogrel, which was followed by dual antiplatelet therapy. A short episode of atrial fibrillation was observed after the procedure.

Figure 1: Initial patient evaluation included thorough anamnesis, physical, ECG that showed ST segment elevations (A) and transthoracic echocardiography (B). The aortic root was measured 26mm, with no signs of underlying aortic disease.
and was addressed with amiodarone infusion followed by oral loading. The patient was discharged in sinus rhythm with amiodarone as a rhythm control therapy. To prevent bleeding, dual antiplatelet therapy (clopidogrel 75 mg and aspirin 75 mg) was prescribed for 1 month. Subsequently, clopidogrel 75 mg was prescribed for 11 more months, and aspirin was replaced by apixaban 2.5 mg bid for long term stroke prevention. No reports of bleeding were found during regular follow up.

Discussion

Aortocoronary dissection is a relatively rare complication. The largest case study to date was published by Shah et al. This condition occurs in less than 0.1% of all patients receiving PCI, and injury to the LM occurs in less than 4% of those cases. [5, 6] The risk factors have been described as age, diabetes mellitus, hypertension, previous coronary artery bypass grafting, concomitant AMI, atherosclerotic burden and any underlying structural weakness of the tunica media [5]. A more recent 20-year retrospective study by Klaudel et al. has evaluated 96 patients with catheter induced coronary dissection, 16 of whom had aortic involvement. The incidence was statistically significantly higher in women, and patients with AMI, chronic kidney disease or prior stroke. An unfavorable origin of the coronary artery, small proximal diameter and ostial atheroma were identified as predisposing factors for coronary dissection [7].

The few reported larger studies have all indicated that the RCA is the culprit vessel in most cases [1, 5, 8, 9]. The treatment of CTO lesions can also be complicated by aortocoronary dissection, particularly when the retrograde approach is used, because of the sometimes challenging crossing of the retrograde guidewire into the true lumen [10].

Figure 2: Coronary angiography, intervention and complication management. A: Evaluation of the left coronary artery revealed proximal subtotal occlusion of the left circumflex artery (arrow); B: Stent placement in the left circumflex coronary artery was followed by (C) aortocoronary dissection with the false lumen starting from the left main coronary artery; D: An emergency decision was taken to stent the left main coronary artery. The markers of the stent (arrows) and the intimal flap between them are visible; E,F: Stent expansion and final result.
The etiology of this complication is complex and not entirely clear. The underlying anatomies of the left and right coronary arteries have been suggested to be sufficiently different to provide a substrate. For example, the greater numbers of concentrically arranged smooth muscle cells and elastic fibers in the tunica media of the LCA make dissection propagation less likely [11]. A histology study by Lopez-Minguez et al. has also reported structural differences between the left and right coronary sinuses and the periostial wall. The left coronary sinus has more smooth muscle cells set within larger amounts of type III collagen, whereas the right coronary sinus has fewer smooth muscle cells in type I collagen. Type I collagen has less tensile strength compared to type III collagen, thus potentially explaining the more frequent retrograde propagation in cases of right coronary artery dissection versus left coronary artery dissections [12].

The precipitating factor for a dissection is usually an iatrogenic mechanism of injury. Catheter and wire manipulations, forceful contrast injection, and balloon and stent expansion may lead to intimal injury [13]. Next the dissection propagates in an uncontrolled fashion, either distally in the coronary artery, thus causing total occlusion, or proximally toward the ascending aorta or contralateral sinus of Valsalva [3, 13]. Deep catheter insertion, particularly during device delivery and removal, malalignment, high systolic-diastolic mobility, respiratory instability and vessel prodding, have also been identified as precipitating factors [7]. The typical smaller size of the right coronary ostium might be a reason for more aggressive catheter manipulation [12]. The Amplatz catheter shape is frequently associated with aorto-coronary dissection with the transradial approach, whereas the Judkins catheter shape is associated with the transfemoral approach [1]. The existing DeBakey and Stanford classifications, although still suitable, are currently supplemented by the Dunning classification, which is currently more broadly recognized. According to the Dunning classification, coronary artery dissections not engaging the aorta are considered class I; those engaging the ipsilateral sinus of Valsalva but less than 40 mm of the ascending aorta are considered class II; and those engaging the ipsilateral sinus of Valsalva and more than 40 mm of the ascending aorta are considered class III [14]. Our patient was classified into class II. A newer and more simplified classification of LM dissections has been proposed by Eschtehardi et al. This classification comprises localized dissections (type I), those extending into the major branches (type II) and those extending into the aortic root (type III) [6]. Our patient would have been classified into type III.

The founders of the Dunning classification have suggested that patients in classes I and II should be indicated for immediate stenting, whereas those in class III should be referred for urgent surgical repair or replacement of the aorta with or without the coronary arteries and the aortic valve [14]. However, more recent retrospective analyses and case series have shown that even class III patients can be successfully managed interventionaly or even conservatively [5, 9, 15, 16]. Furthermore, the in-hospital mortality rate among surgically managed patients is high, at 38.5% [17].

Prevention strategies usually include vigilance when working with the described patient populations and catheter shapes. Device manipulations should be succinct and precise, to decrease traction and the chances of intimal disruption. Catheter coaxiality and proper intubation should be maintained at all times, particularly with signs of proximal or ostial disease. Contrast should never be injected in the case of a wedged catheter [7, 18].

Specific aspects are critical in the treatment of aortocoronary dissection; rapid placement of a coronary stent is usually sufficient to seal the intimal flap and thus limit retrograde propagation into the ascending aorta. Sometimes, when the dissection also engages a bifurcation, more complex bailout techniques must be used, such as T-stenting and the small protrusion technique described by Lao et al. in a similar case [19]. Extensive contrast use during the procedure can increase propagation of the false lumen and therefore should be limited. Hence, the use of newer techniques such as optical coherence tomography or intravascular ultrasound is highly advocated for and has achieved very favorable outcomes. When these modalities are unavailable, the procedure can be performed under plain fluoroscopy [20]. In retrograde revascularization of CTO lesions this complication can be evaded by paying meticulous care not to advance the guidewire into the sub aortic space. Re-entry into the true lumen should be kept as distal of the ostium of the target vessel as possible [10].
Reports have recommended multidetector computed tomography as the imaging modality of choice for follow-up after aortocoronary dissection. This method has high spatial resolution and potential for 3D imaging, and is operator independent, unlike transesophageal echocardiography, for example. The proposed protocols for follow-up MDCT include time points immediately after the procedure, 48 hours, 1 week and 1 month [15].

In line with previous reports of LM dissections, we chose a bail-out stenting management strategy [19]. Newer technologies such as intravascular ultrasound can aid in identifying the initial entry point of the dissection, and the presence of thrombus or intramural hematoma, and in ensuring proper stent placement [19, 13]. However, we demonstrated that management of this complication is also feasible with core techniques. Immediate stenting is generally the treatment of choice, because it often resolves the problem. This treatment should always be attempted, because it does not make surgical repair impossible and in fact can provide hemodynamic stability by ensuring the patency of the left anterior descending artery and LCx while the patient is prepared for surgery [3].

**Ethical Statement**

The authors declare that patient consent was obtained for the publication of this specific clinical case. All reasonable steps to maintain confidentiality and anonymization have been taken.

**Conflict of Interest**

The authors declare no conflicts of interest.

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