An Experience of Catheter-Induced Aortocoronary Dissection Complicated by Subtle Coronary Perforation

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Aortocoronary dissection and coronary perforation are rare complications of percutaneous coronary intervention (PCI). We present a 68-year-old man with aortocoronary dissection involving the right coronary artery (RCA) and ascending aorta during PCI. We tried to seal off the entry site by using conventional stenting but failed. After reviewing angiograms, a subtle leakage of contrast into the pericardial space before stenting led us to the diagnosis of coronary perforation. Finally, we successfully treated this complication by deploying a covered stent. This case report reminds us that coronary perforation could be missed if there is only subtle contrast jet in the angiogram. Prompt treatment with conventional stenting may be harmful for coronary perforation. Detailed review of angiograms before management of coronary dissection is mandatory.

Key Words: Aortocoronary dissection • Subtle coronary perforation • Covered stent • Percutaneous coronary intervention

INTRODUCTION

Procedure-induced aortocoronary dissection or perforation was rarely reported, the incidence rate varies between 0% and 0.02% for diagnostic procedure and 0.03% and 0.15% for angioplasty procedures.1-4 Sometimes, it’s difficult to discern between coronary perforation and dissection, but unnoticed, minimal pericardial effusion may be an important clue. The management for coronary dissection and perforation is different but adequate, rapid recognition of these complications is important and life saving. Here we reported a patient who developed a catheter-induced aortocoronary dissection complicated by subtle coronary perforation during coronary intervention.

CASE REPORT

A 68-year-old man presenting with effort chest tightness for months and unstable angina was admitted to our hospital. He had diabetes mellitus, hyperlipidemia and essential hypertension as coronary risk factors. The coronary angiography revealed triple-vessel disease; however, he refused surgical treatment. Percutaneous coronary intervention (PCI) was performed for the culprit lesion of the left anterior descending artery the first time. Optimal medications were given after procedure, including both aspirin and clopidogrel. Three months later, he was admitted for scheduled PCI of the distal right coronary artery (RCA), which had chronic total occlusion lesion. We used a 6Fr Amplatz left 2 (AL2) short tip guiding catheter via the left radial artery. No evidence of ulcerous or dissected atheromatous plaque was observed in the ostium or proximal segment of the RCA. After intra-coronary injection of heparin acetate (6000 units)
and isosorbide dinitrate (1 mg), an Asahi-intermediate wire (Asahi Intecc. Co., Ltd., Aichi, Japan) with a Progreat microcatheter (Terumo Corp., Tokyo, Japan) was advanced to the total occlusion of the RCA but failed. Therefore, we attempted a Miracle 4.5-g guide-wire (Asahi Intecc. Co., Ltd.), but it seemed to enter a false lumen of distal RCA. At this moment, the angio-
gram showed severe spiral dissection involving the prox-
imal RCA and the wall of aortic root with compromised coronary flow (Figure 1A). The patient complained of mild chest tightness and developed tachycardia without obvious drop of blood pressure. In order to maintain blood flow to the RCA and reduce myocardial ischemia, we decided to seal off the entry site of dissection with stenting. As the dissection could be caused by the man-
ipulation of the 6 Fr AL 2 short tip guiding catheter and the rigid wire, we replaced it by a 6 Fr JR 5 guiding catheter and recrossed the dissected segment of RCA via Runthrough NS floppy guide wire (Terumo corporation) to avoid further trauma. A 3.0 × 18-mm Driver stent (Medtronic Inc., Minneapolis, MN, USA) was deployed in proximal RCA with the proximal end outside the ostium. After stenting, intravascular ultrasound (IVUS) demonstrated that the RCA’s vessel diameter was 4.0-4.5 mm, with intramural hematoma (Figure 1B). Therefore, post-stent balloon dilation was performed with a 4.0 × 12-mm Quantum non-compliance balloon (Boston Scientific Scimed Inc.) at 20 atm for appropriate apposition of the stent. But subsequent coronary angiography showed incomplete seal-off of the dissection (extension of contrast into aortic cusp, ascending aorta wall). After reviewing previous images, leakage of contrast into the pericardial space, compatible with coronary perforation of Ellis classification grade 2, was found before replacing the guiding catheter and guidewire (Figure 1C). So we changed our treatment strategy from aorto-coronary dissection to aorto-coronary perforation. After reversal of heparin with protamine sulfate 30 mg intravenously, we decided to deploy a 3.5 × 19-mm covered stent (Jostent, coronary stent graft ) (Jomed International, Germany), the best optimal stent size available in our facility. Post-dilation was performed using a Quantum 4.0 × 12 mm balloon at 22 atm (Figure 1D).

Subsequent coronary angiography showed no further leakage or extension of contrast into the aortic cusp, ascending aorta wall, or pericardial cavity. Transthoracic echocardiograms obtained during the procedure, imme-
diately after the procedure, and one and 2 days later demonstrated no pericardial effusion or extension of the aortic dissection. We therefore maintained dual antiplatelet therapy. The patient was discharged two days later and remained free of symptoms until now.

Figure 1A. Left anterior oblique cranial view. Large antegrade and retrograde dissection involving RCA, aortic root and ascending aorta is shown.

Figure 1B. Image from intravascular ultrasound. After deploying the Driver stent, we inserted IVUS, which showed adequate apposition of stent struts and intramural hematoma.
DISCUSSION

Procedure-induced aortocoronary dissection or perforation is rarely reported. Risk factors for coronary perforation include older age, female gender, vessels with complex coronary anatomy (severe calcification, tortuosity of the vessel, extreme angulation, bifurcation, chronic total occlusion, ostial lesion, ACC/AHA type B or C lesion), prior CABG, high balloon-stent ratio, high inflation pressure and distal location of guide wire, and use of devices like hydrophilic coated wire, stiff wire, cutting balloon, and atheroablative devices.  

Although our patient had some of the above mentioned risk factors for predisposed further perforation (ACC/AHA type C lesion, use of high pressure and stiff wire), the etiology of aortocoronary dissection and perforation was most probably caused by manipulation of the Amplatz guiding catheter. The relationship between the guiding catheter and incidence of aortocoronary dissection and perforation was most probably caused by manipulation of the Amplatz guiding catheter. The relationship between the guiding catheter and incidence of aortocoronary perforation is controversial; some authors consider catheters like IMA, multi-purpose and Amplatz may cause dissections during catheter manipulation. However, some reports demonstrated that JR catheters were more frequently involved in dissection.  

Awareness of the potential risk, using appropriately sized and shaped catheters, and avoiding forceful contrast injection if the pressure is damped may aid in prevention, rapid recognition of this complication, and therefore potentially improve the speed with which definitive therapy may be instituted.  

Ellis et al. defined angiographic classification for coronary perforation with increased incidence of pericardial effusion or cardiac tamponade in type II or III classification or receiving GP IIb/IIIa antagonists. The incidence of coronary perforation during PCI in the new device era characterized by high usage of secondary devices and GP IIb/IIIa platelet antagonist remains low, ranges from 0.1% to 0.4%. The most important cause is iatrogenic, more frequently with debulking devices such as directional coronary atherectomy and rotational atherectomy, than with the use of non-debulking procedures such as balloononing and coronary stenting.

It’s worth mentioning that the incidence of guidewire-related perforation (crossing lesion, distal wire perforation and wire fracture) may be underestimated because sometimes it is unrecognized and self-limited. It has been reported that perforations occurred with the use of hydrophilic guidewires in 50% of patients, with the use of intermedius and standard guidewires in 14%, with the use of floppy tip wires in 29% and with the use of a Rota-floppy guidewire in 7%. Perforation is likely to
occur when intravascular ultrasound (IVUS) is used. The main reason is an indirect interpretation of the information provided by IVUS and the usage of compliant oversized balloons to maximize the stent lumen area.5

Type I and type II coronary perforations are usually confirmed by retrospective review of the angiogram, especially when hemodynamic status is unstable. Type III coronary perforation is often recognized by contrast extravasation, and tamponade is usually diagnosed by echocardiography or occasionally by detecting the tamponade physiology. Fluoroscopy may reveal immobile heart border when cardiac tamponade has occurred. We should be aware of coronary perforation if hypotension occurs post PCI because undetected pericardial effusion may progress to cardiac tamponade in 8-24 hours.12

There are no evidence-based guidelines to assist the operator in treating catheter-induced aorto-coronary dissection or perforation. Several factors influence the selection of treatment strategies, which include conservative management, stenting, and surgical operation. Some authors mentioned that in conditions like extensions of aortic dissection more than 40 mm from the coronary cusp, dissection-related acute myocardial infarction, acute aortic regurgitation, or cardiac tamponade, surgery should be performed promptly.5 Only a very small percentage of aortocoronary dissection like these would be sealed off spontaneously.

Prolonged balloon inflation, with a balloon, size-to-artery size ratio of~1.0, positioned over the site of extravasation for 10-15 minutes and a perfusion balloon is an option to avoid myocardial ischemia during balloon inflation. Reversal of anticoagulation may be warranted, such as intravenous protamine sulfate for heparin reversal achieving a partial thromboplastin time less than 60 seconds or an activated coagulation time less than 150 seconds, and platelet transfusion for abxicimab, but not for tirofiban or eptifibatide. The vessel size and the severity of coronary perforation may influence in the further conservative management. In perforation types II and III, besides the above management, a polytetrafluoroethylene (PTFE) covered stent can be deployed if the vessel diameter is equal or greater than 2 mm.8 For smaller coronary artery, microcoil/gelfoam embolization has been developed.13 Surgical treatment should always be performed promptly if conservative management fails or the clinical situation warrants.8,13,14

A covered stent (also known as coronary stent graft) has a PTFE stent is easy and rapid to deploy, it may prevent catastrophic consequences and avoid prolonged balloon inflation on the injured vessel which may not guarantee sealing off the entry site and may increase the procedure time and thrombosis. However, there are still major concerns regarding covered stents: restenosis rate (14 to 39%), the side branch occlusion rate (18%), stent thrombosis (5.5%) compared to the conventional stent (3.6%) and making prolonged use of antiplatelet medications necessary.11,13

In conclusion, aortocoronary dissection or perforation during PCI is a rare complication but life-threatening. Careful selection and manipulation of catheters and paying more attention to high-risk patients are important to avoid these complications. Prompt treatment with conventional stenting for coronary dissection is essential and lifesaving, but may be harmful for coronary perforation. Coronary perforation could be missed if there is only subtle contrast jet in the angiogram. Detailed review of the angiograms when complications occur is mandatory.

REFERENCES


