Successful conservative management of Class III iatrogenic aortic dissection

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Iatrogenic aortic dissection (IAD) is a rare complication of percutaneous coronary intervention (PCI)¹−⁴ with an estimated incidence of 0.02%–0.04%.¹,⁵ IAD occurs more frequently in the setting of urgent PCI for acute myocardial infarction (AMI) (0.19%) than for elective procedures (0.01%).¹ The mortality risk associated with IAD is high (16%) and is comparable to that for spontaneous aortic dissection (16%).³,⁴ Urgent surgery is the treatment of choice¹,⁵ for extensive dissection or hemodynamic instability. In this report, we present a case of severe IAD that was managed conservatively with good clinical outcome.

An 81-year-old Asian woman with history of hypertension and hyperlipidemia underwent elective PCI for stable angina. The patient was on long-term aspirin (100 mg) and was loaded with clopidogrel (300 mg) the day before PCI. Coronary angiography was performed using a right radial artery approach showed 30% stenosis in the left main (LM) stem, 80% stenosis in the distal left circumflex (LCx) artery, 90% stenosis in the proximal left anterior descending (LAD) artery and chronic total occlusion (CTO) in the middle right coronary artery (RCA) which was filled via collateral arteries from the left coronary arteries (Figure 1). The patient refused coronary artery bypass grafting and opted for multi-vessel PCI.

A 6 French Ikari left (IL) 3.5 guiding catheter (Terumo Corporation, Tokyo, Japan) was used to engage the RCA with difficulty and multiple CTO guidewires were required to cross the lesion. RCA CTO was finally crossed with a Gaia second 0.014” guidewire (ASAHI INTECC CO., LTD., Japan) with Turnpike micro-catheter (Vascular Solutions, Inc, Minneapolis, USA) support. Subsequent balloon angioplasty was performed with a 2.0 mm × 10 mm semi-compliant balloon up to 6 atm. Contrast staining was noted in the aortic root after balloon angioplasty. Non-selective angiogram showed a small (5 mm) ascending aortic dissection originating from the RCA ostium.

Emergent stenting was performed from the middle to ostial RCA with a 2.5 mm x 40 mm and 3.0 mm x 26 mm Osirio drug-eluting stents (Biotronik AG, Bulach, Switzerland). The extent of dissection worsened after stenting with a Class III aorto-coronary dissection (contrast extending > 40 mm up the aortic wall) was noted (Figure 2). Intravascular ultrasound (IVUS) assessment showed dissection flaps at the ostium of RCA (Figure 3). Therefore, another 3.5 mm x 18 mm Osirio drug eluting stent was deployed slightly protruding into the aorta to completely cover the RCA ostium. The final angiogram showed successful stenting of the RCA CTO lesion but still persistent contrast leakage into the aortic root (Figure 4).

After stenting, the patient complained of increased chest pain and deteriorated hemodynamically into cardiogenic shock with systolic blood pressure (SBP) of 70 mmHg. Transthoracic echocardiography showed 1.5 cm pericardial effusion with early diastolic right ventricular collapse suggestive of cardiac tamponade. Protamine sulfate was administered for complete reversal of anticoagulation. Pericardiotomies was performed under fluoroscopy guidance from subxiphoid approach, draining 800 mL of fresh blood. Cardiothoracic surgical (CTS) team was urgently consulted for possible open repair of the aortic root and ascending aorta with re-implantation of the coronary arteries. In view of her advanced age and poor prognosis, conservative treatment was recommended by the CTS team.

No further output from the pericardial drain was noted and a repeated echocardiogram revealed a static amount of 0.5 cm pericardial effusion. Patient was titrated on low-dose inotrope with SBP maintained at around 80 mmHg intentionally in the catheterization laboratory. The patient was then transferred to coronary care unit (CCU) for close monitoring.
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Figure 1. Left and right anterior oblique view. (A): Left anterior oblique view. Angiogram showed right coronary artery chronic total occlusion segment (arrows). (B): Right anterior oblique view. Angiogram showed right coronary artery chronic total occlusion segment (arrows) with collateral arteries from the left coronary artery (*).

Figure 2. Class III aortocoronary dissection with contrast extends to > 40 mm up the aortic wall. Stent (arrow) was protruded into the aorta to seal dissection flap in the right coronary artery ostium.

During the stay in CCU, aspirin and clopidogrel were withheld. There were no dynamic ECG changes or significant rise in cardiac enzymes for 72 hours. Transthoracic echocardiography was repeated 24 hours later showed static 0.5 cm of pericardial effusion without any suction. The left ventricular ejection fraction was 55 percent. The pericardial drain was removed 48 hours after the procedure in view of no further drain output. Aspirin was restarted 48 hours after the procedure and clopidogrel was resumed on post-procedure day 4. She was discharged on post-procedure day 10. At three-month clinic follow-up, the patient remained free of chest pain.

Figure 3. Intravascular ultrasound assessment showed dissection flaps at the ostium of right coronary artery (RCA). RCA: right coronary artery.

Aortic dissection can be iatrogenic.[6] It can become immediately life-threatening via a number of mechanisms, including (1) hemorrhage into the pericardium resulting in cardiac tamponade and hemodynamic collapse; (2) occlusion of the contralateral coronary ostium (e.g. occlusion of the LM coronary artery during PCI to the RCA); (3) occlusion of other aortic arch vessels, resulting in cerebrovascular accidents; or (4) propagation of the dissection into the descending aorta. The occurrence or potential for these complications may mandate surgical intervention, which carries a mortality risk of up to 25%. [6]

IAD may involve distinct mechanisms from spontaneous dissections due to atherosclerosis or connective tissue dis-
orders. The involvement of occluding lesions at the RCA, usage of stiff wires and catheters wedged or in a non-coaxial position relative to the vessel wall were the likely causes of IAD in this case. Dunning and colleagues\(^{[1–4]}\) have classified aorto-coronary dissection based on the degree of aortic involvement: Class I, contrast staining involves only the coronary cusp; Class II, contrast extends up the aortic wall to < 40 mm; and Class III, contrast extends to > 40 mm up the aortic wall. This classification is mainly used for risk stratification with progressively worse prognosis with higher IAD classes. The optimal management for IAD is unclear and more controversial compared to spontaneous Type A aortic dissection, for which emergent surgical intervention is recommended. Some recommend that cases of localized aorto-coronary dissection not complicated by ischemia or hemodynamic instability can be treated with intracoronary stenting or conservative management. By contrast, if there is ischemia in any of the aortic branches, or if there is extensive dissection or hemodynamic instability, as in our case, urgent surgery is recommended.\(^{[1–5]}\)

In this case, several management steps were crucial. Intracoronary stenting could seal the dissection flap. Prompt reversal of anticoagulation could lead to thrombosis and spontaneous sealing of the false lumen. Early recognition of IAD and hemodynamic instability could guide prompt diagnosis of cardiac tamponade. By maintaining a low systolic blood pressure around 80–90 mmHg could potentially prevent further propagation of the dissection that occurs with pulsatile flow. The anti-platelet regime has to be individualized by balancing the risk of bleeding and stent thrombosis.

Our case illustrates conservative management of a class III iatrogenic IAD after PCI with good clinical outcome.

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