IVUS-guided Percutaneous Coronary Intervention Helps in Salvaging Catastrophic Iatrogenic Aortic Dissection Extending from Left Main Coronary Artery

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Abstract

Iatrogenic aortic dissection could be a serious complication during percutaneous coronary intervention. Prompt identification and proper management make a great difference regarding the clinical outcome. To manage this catastrophic complication, a dedicate angiographic reading matters, and a comprehensive CT scan is suggested to define the extension, immediate treatment effect and resolution of vascular complication. The literature regarding iatrogenic aortic dissection was reviewed.

Keywords: Iatrogenic aortic dissection, percutaneous coronary intervention, coronary artery disease, intravascular ultrasound

Introduction

Iatrogenic aortic dissection causing from coronary artery dissection was a well-known catastrophic and potentially fatal complication during coronary angiogram acquisition and percutaneous coronary intervention. According to the literature, the incidence was around 0.02-0.07%.[1-3] It was mostly caused by the manipulation of guiding catheter, guidewire, balloon and forceful contrast injection. It was more frequently seen during the intervention of RCA.[3,4] Here we shared a 65-year-old female case of unexpected iatrogenic aortic dissection during percutaneous coronary intervention.

Case report

This was a 65-year-old female patient complaining of intermittent chest tightness for one year. Her cardiovascular risk factors consisted of age, hypertension, and family history of premature coronary artery disease (one of her brothers died of acute myocardial infarction).

Her electrocardiogram showed a normal sinus rhythm with mild T wave inversion in V1 to V3 (Figure 1A). A thallium myocardial perfusion scan showed significant stress-induced ischemia at apical myocardium (mixed with subendocardial scar at apical lateral wall), inferolateral, and mid to basal inferior walls (Figure 1B).

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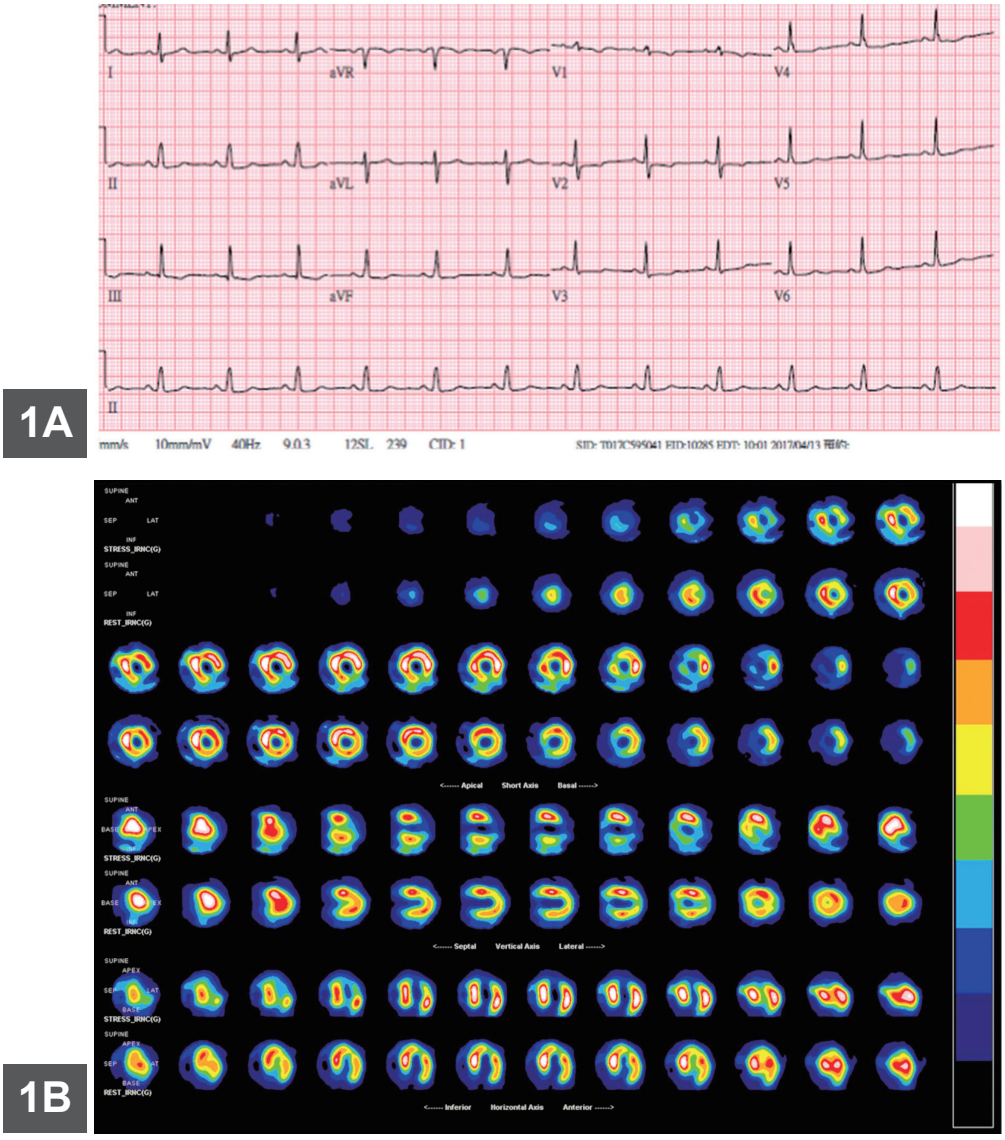

Figure 1. Noninvasive studies of coronary artery disease. A. The baseline electrocardiogram shows normal sinus rhythm without significant ST and T wave change. B. The thallium myocardial perfusion scan shows significant stress-induced ischemia at apical myocardium (mixed with subendocardial scar at apical lateral wall), inferolateral, and mid to basal inferior walls.

A coronary angiogram was obtained and revealed left main and two-vessel disease. The left main (LM) coronary artery shaft was calcified and 50% stenotic. The left anterior descending (LAD) artery was 70% stenotic at proximal segment and 99% stenotic at middle segment. The left circumflex artery (LCX) was patent with collateral to right coronary artery (RCA) territory. The RCA was 90% stenotic at proximal segment and 90% stenotic at distal segment, with collateral to LAD territory (Figure 2A-2D).

Coronary artery bypass surgery was advised, but the patient opted for percutaneous coronary intervention instead. The strategy was to fix the LAD first. The procedure was conducted via right common femoral artery with a 7-French

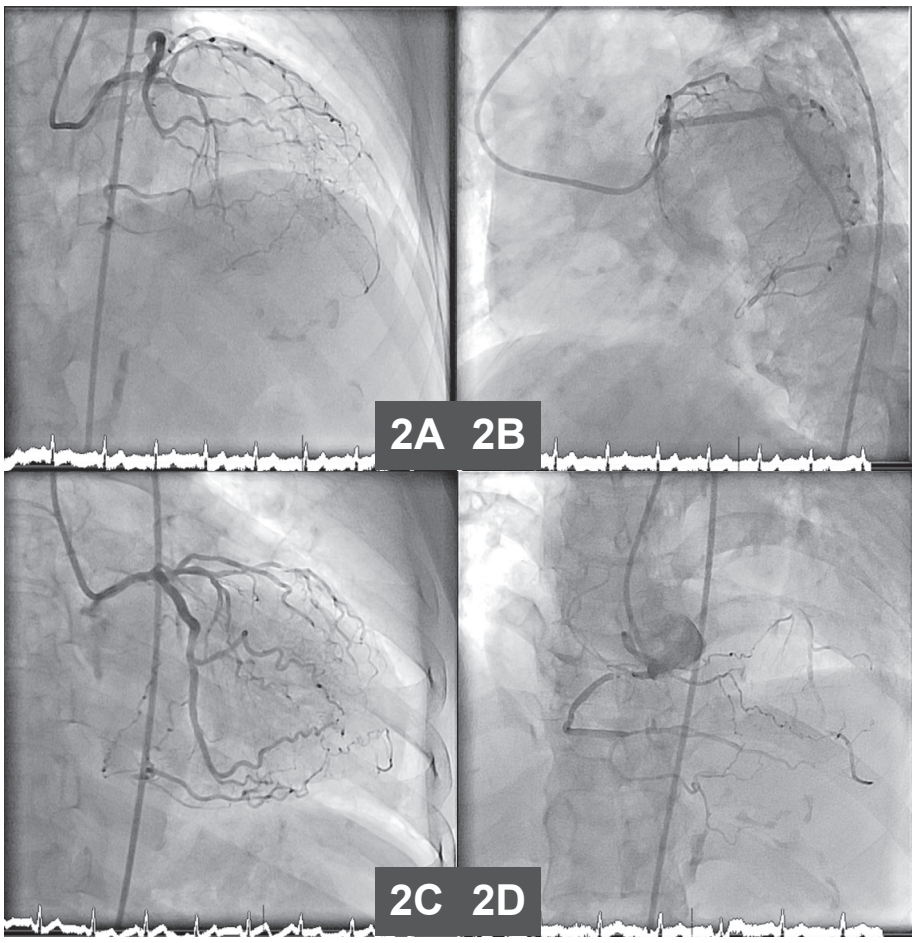

Figure 2. The diagnostic coronary angiogram in this index procedure. A. The left coronary artery (LCA) right anterior oblique (RAO) cranial view shows the LAD is 70% stenotic at proximal segment and 99% stenotic at middle segment. B. The LCA left anterior oblique (LAO) caudal view shows the left main (LM) coronary artery shaft is calcified and 50% stenotic. C. The LCA RAO caudal view shows the left circumflex artery (LCX) is patent with collateral to right coronary artery (RCA) territory. D. The RCA anterior-posterior cranial view shows the RCA is 90% stenotic at proximal segment and 90% stenotic at distal segment, with collateral to LAD territory.

Medtronic EBU 3.5 guiding catheter. An ASAHI-Fielder XT-A wire, supported by a 130 cm Terumo Finecross microcatheter, crossed the middle LAD critical lesion smoothly. An intravascular ultrasound (IVUS) pullback was obtained to determine lesion length and vessel size. The middle LAD lesion was predilated by a 2.5 mm x 1.5 mm semi-compliant balloon. Long dissection was noted after pre-dilation, and a 2.5 mm x 48 mm drug-eluting stent (DES) was delivered to the proximal-middle LAD smoothly. However, right after the contrast injection prior to stent deployment, a horrible image presented in our face with sudden circulatory collapse and subsequent seizure attack (Figure 3A-3B).

It was an iatrogenic retrograde aortic dissection from LM coronary artery accompanied with a ventricular fibrillation and subsequent cerebral hypoperfusion. Electric cardioversion with 200 joule energy was performed twice, and the rhythm returned to normal sinus rhythm. Hypotension occurred and the blood pressure

was supported by dopamine. Fortunately, her consciousness recovered and her vital signs were temporarily stabilized under inotrope support.

The left coronary cusp and ascending aorta were involved. This was a Dunning class 2-3 iatrogenic aortic dissection.[1] Either surgical repair or direct stenting could be considered. PCI was chosen to save the time. Because of severe dissection, further contrast injection should be avoided. IVUS-guidance was crucial to adequately salvage this complication. IVUS was used first to evaluate the extension of dissection. The dissection extended from middle LAD to LM ostium. The LM ostium position was marked by the IVUS probe on the fluoroscopy image (Figure 4A). Another workhorse wire was introduced into LCX by the assistance of a double-lumen microcatheter to ensure that the wire was in the true lumen. The previous 2.5 mm x 48 mm DES was deployed at proximal to middle LAD (Figure 4B). Another 3.0 mm x 33 mm DES was deployed from proximal LAD to LM ostium guided by the previous IVUS probe mark on fluoroscopy image (Figure 4C).

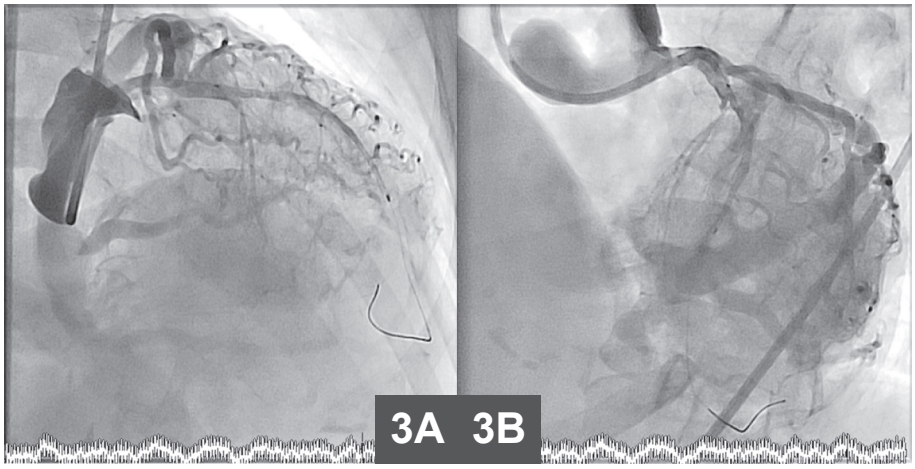

Figure 3. The angiogram of iatrogenic LM coronary artery retrograde aortic dissection. A. LCA RAO cranial projection. B. LCA LAO cranial projection.

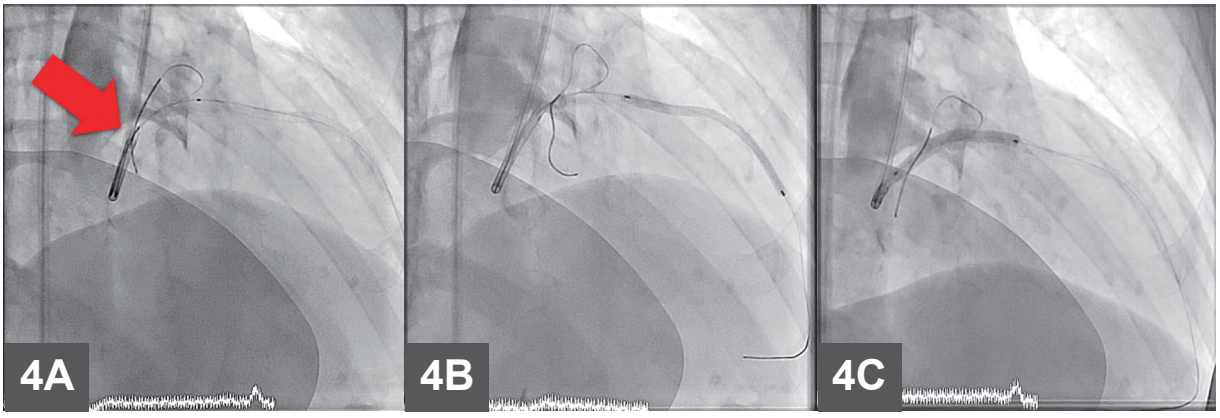

Figure 4. The process of IVUS-guided PCI. A. The LM ostium mark by IVUS probe. B. A 2.5 mm x 48 mm DES was deployed at proximal to middle LAD. C. Another 3.0 mm x 33 mm DES was deployed from proximal LAD to LM ostium guided by the previous IVUS probe mark.

A kissing balloon technique was performed to the LM bifurcation with a 3.5 mm x 15 mm non-compliant balloon in LM-LAD and a 2.5 mm x 15 mm non-compliant balloon in LM-LCX. A proximal optimization technique was performed to the LM with a 3.5 mm x 15 mm non-compliant balloon. The angiographic result showed contrast retention in the ascending aorta without further progression. The inlet of dissection was successfully sealed (Figure 5A-5B).

The patient was sent to the coronary care unit and dopamine was discontinued on the next day. The immediate chest computed tomography (CT) after the procedure showed contrast retention in the ascending aorta and pericardium. (Figure 6A-6B) The post-procedural course was uneventful, and the patient was discharged home two days after the procedure.

Three months after the index procedure, a follow-up chest CT scan showed completely sealed aortic dissection. A follow-up coronary angiogram showed patent left coronary arteries and complete resolution of previous aortic dissection (Figure 7A-7B). The RCA became totally occluded (Figure 7C) and was revascularized successfully with implantation of two drug-eluting stents (Figure 7D). Complete revascularization was finally achieved.

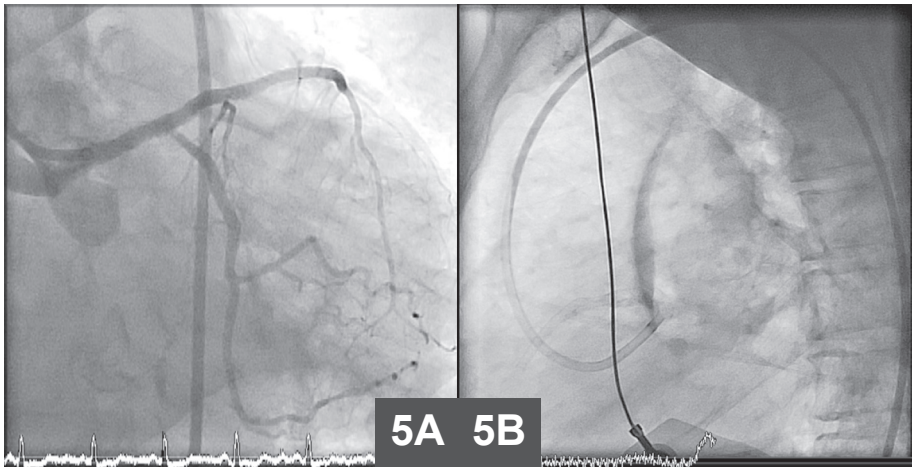

Figure 5. The inlet of dissection was successfully sealed. A. The final LCA angiogram shows satisfactory LAD stenting result. B. The final ascending aorta fluoroscopy image shows contrast retention in the ascending aorta without further progression.

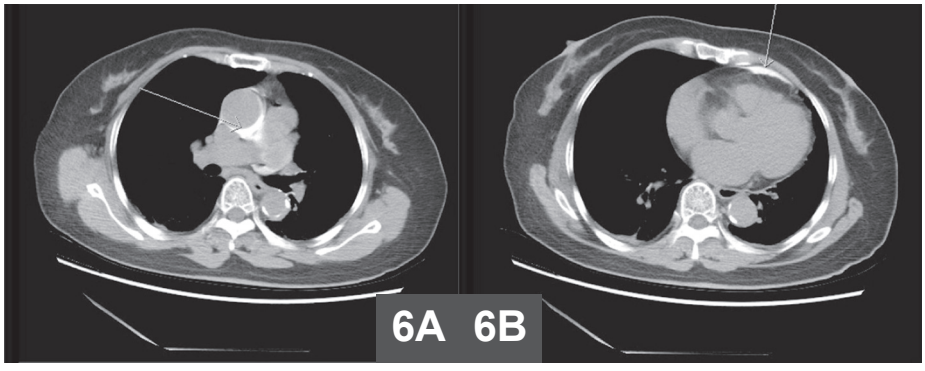

Figure 6. The immediate chest CT result after the index PCI. A. Contrast retention at ascending aorta. B. Contrast retention in the pericardium.

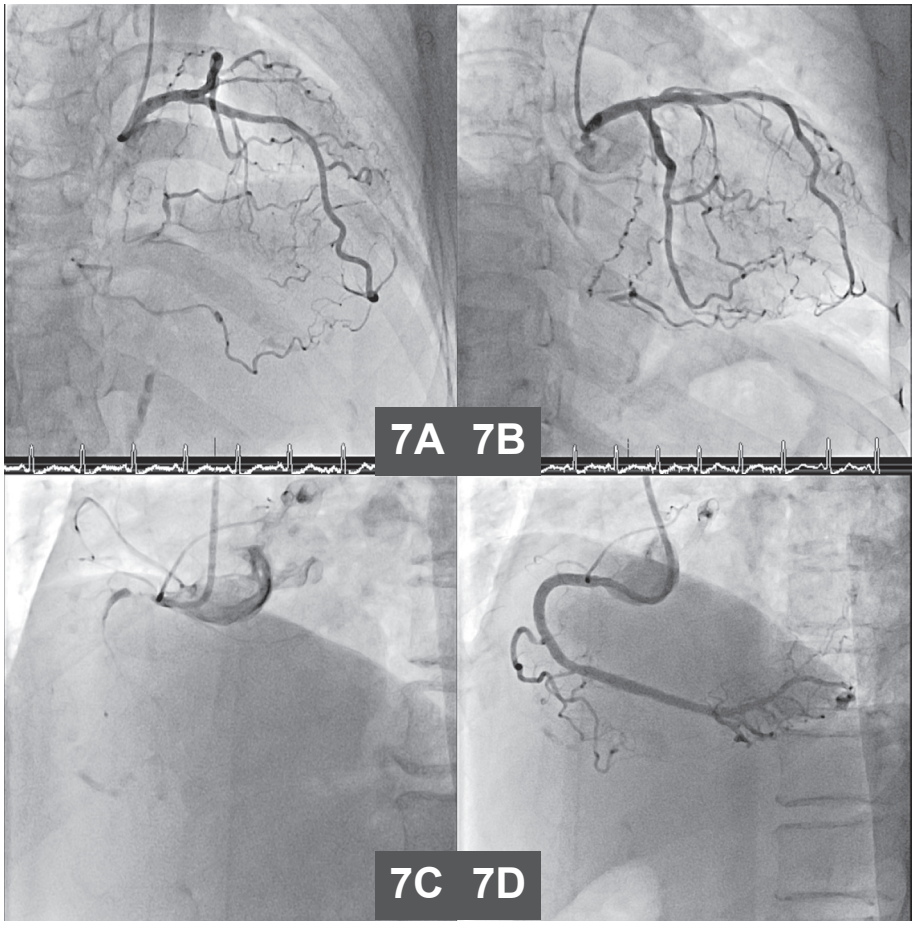

Figure 7. Follow-up coronary angiogram and staged percutaneous coronary intervention of the right coronary artery 3 months after the index procedure. A. The LCA RAO cranial view shows good LAD stenting result without ISR. B. The LCA RAO caudal view shows patent left coronary arteries. C. The RCA LAO 60 degree view shows a totally occluded RCA. D. The final result of RCA PCI.

Discussion

Iatrogenic aortic dissection causing from coronary artery dissection was a well-known catastrophic and potentially fatal complication during coronary angiogram acquisition and percutaneous coronary intervention. However, due to low incidence and different causes of iatrogenic aortic dissection, it was difficult to give universal recommendations regarding optimal management. Our case was saved with a proper and prompt management. Again, our case demonstrated that a dedicate angiographic reading, a prompt react and a CT scan were of much help regarding in saving this catastrophic complication. A comprehensive CT scan is suggested to define the extension, immediate treatment effect and resolution of vascular complication.[5] An intravascular ultrasound played a significant role in our case also.

In an European database (Registry on Aortic Iatrogenic dissection [RAID]) reported by Núñez-Gil et al., a total of 74 patients with iatrogenic aortic dissection resulting from interventional procedures were identified.[3] Regarding the cause of dissection, engaging coronary vessels accounted for 72 patients (97.2%) of these events (42 and

30 patients for RCA and LCA, respectively). The dissections were causes by catheters in 68 patients (91.8%) and by 0.035-inch wires in 4 patients. Among those culprit catheters causing iatrogenic aortic dissection, 70.3% were guiding catheters and 90.5% were 6F in size. Judkins (42.9%) was the most frequent curve in cases via femoral access. For radial access, Amplatz (38.9%) was the most frequent one.[3]

In 2000, Dunning et al. tried to guide the selection of treatment modalities by classifying the disease entity into three categories, including Dunning class 1 (involving the ipsilateral coronary cusp), class 2 (involving cusp and extending up the aorta < 40 mm) and class 3 (involving cusp and extending up the aorta > 40 mm).[1] To our best knowledge, this was the only published evidence in literature focusing on disease severity classification and treatment strategies. In this retrospective review of 9 cases, the Dunning class 1 dissection could be managed by conservative treatment. The Dunning class 2 dissection could be managed by either conservative treatment or stenting. The Dunning class 3 dissection was mostly managed by an immediate surgery. However, in the modern era, some interventionalists thought direct bailout stenting could help even in the Dunning class 3 dissection. The median 5-year follow-up result of the RAID registry even suggested a worse outcome in patients receiving surgical interventions, considering the complexity of surgery involving aorta and coronary vessels and the concurrent use of anti-thrombotic agents in those patients.[3]

The optimal management of aortic root dissection remains a debate because of low incidence and difficulty conducting a randomized study in this patient group. Primary coronary stenting to seal the flap entrance seemed successful in various case reports, even with the Dunning class 3 disease. Prompt recognition and a rational treatment strategy considering the hemodynamics and anatomy are essential to successfully save patients.

The cause of iatrogenic aortic dissection in our patient was thought to be forceful contrast injection while there was an acute angle between the guiding catheter and the left main coronary artery. This should be avoided in our daily practice. We successfully salvaged the complication by IVUS-guided PCI, which avoided further contrast injection before the dissection was sealed. A double-lumen microcatheter-assisted side-branch wiring in the setting of severe coronary dissection was also crucial in our case.

Conclusion

The optimal management of iatrogenic aortic root dissection from coronary arteries remains a debate. Prompt recognition and rational management are essential to save patients. IVUS-guided PCI helps to evaluate the extension of dissection and to reduce the possibility of false lumen propagation caused by further contrast injection.

Disclosure

There was no conflict of interest for this report among the authors.

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