Reviews

Aortic and Coronary Artery Dissection During Percutaneous Coronary Intervention: A Case Report and Review Article

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ABSTRACT

Coronary artery dissection is an uncommon but potentially serious complication in percutaneous coronary interventions. We treated a 53-year-old female patient with right coronary spasm, which was misdiagnosed as a coronary lesion. The coronary spasm resolved with nitrate administration, but proximal coronary dissection developed during angiography. It progressed anterograde and led to inferior myocardial infarction and severe hemodynamic instability due to right coronary artery occlusion. Percutaneous intervention failed, and it was determined that coronary dissection progressed retrograde, across the truncus of the aorta. Ascending aortic grafting and coronary bypass surgery were performed. The patient recovered and was discharged after 10 days. Close cardiothoracic observation is mandatory in patient evaluation and management.

Introduction

Incidence of catheter-induced coronary artery spasm is 0.2%–3% during coronary intervention.[1–3] Optimization of medical therapy, especially use of vasodilators, has been widely accepted as the initial approach to management. Coronary spasm closely resembles a fixed atherosclerotic lesion. Coronary dissection and its progression into arcus aorta is an extremely rare but potentially life-threatening complication of percutaneous coronary intervention (PCI). The cumulative incidence of catheter-induced dissection with retrograde progression to the aorta has been estimated at approximately 0.008% in diagnostic coronary angiography.[4] In this case report, we present a patient with coronary spasm and coronary dissection with both anterograde and retrograde progression due to the PCI.

Case Report

A 53-year-old female complaining of chest pain was admitted to one of our outpatient cardiology clinics. She had hypertension and smoking as coronary risk factors. After a positive exercise stress test, coronary angiography was performed. The angiography indicated critical luminal stenosis (50%–60%) at the midsection of the right coronary artery (RCA; Figure 1A). A PCI was planned, but the patient was discharged at her own request.

Three days later, the patient was admitted to a different cardiology clinic and coronary angiography was again performed. In left and right cranial imaging in the angiogram, coronary spasm, but no significant lesion, was observed in the mid RCA. The coronary spasm disappeared after nitrate administration (Figure 1B). The previous lesion was accepted as coronary spasm, so the patient was admitted to the coronary service without any intervention. But, the next morning, she experienced sudden-onset, squeezing, precordial chest pain, and again was taken to the catheterization laboratory. The angiogram showed total occlusion of the distal RCA (Figure 2A); the possibility of coronary spasm was considered and then distal flow was regained after nitrate administration (Figure 2B). However, it was noticed that coronary dissection had occurred in the proximal RCA, which had caused total occlusion in the proximal RCA (Figure 2C,D). Cardiac and respiratory arrest occurred. The patient's rhythm was ventricular fibrillation, and defibrillation was performed. The electrocardiogram showed ST-segment elevations in leads DII, DIII, and aVF; ST depressions in V_1 and V_3; and complete atrioventricular block. A temporary cardiac pacemaker was implanted. The patient had lost consciousness and she was intubated. Total occlusion still persisted in the proximal RCA (Figure 3A). A 0.014-in. floppy guidewire was advanced to the distal section of the RCA. Intracoronary nitrate was administered

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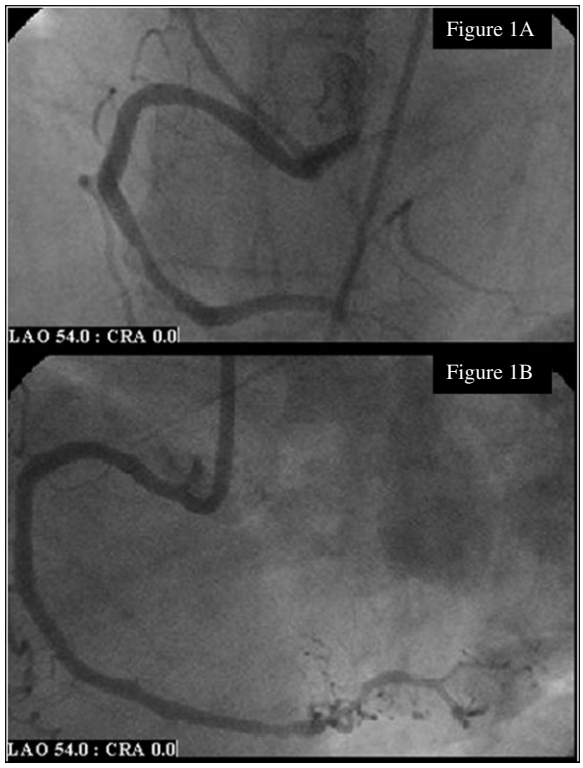


Figure 1. (A) Coronary spasm in midsection of RCA and (B) disappearance after nitrate administration. Abbreviations: RCA, right coronary artery.

for the coronary spasm (Figure 3B). Coronary distal flow was impaired by a newly developed severe diffuse mid-RCA lesion. The RCA midsection was dilated with a 2.5 × 20-mm balloon (Figure 3C). After balloon dilation and repeated intracoronary nitrate, no reflow was observed in the distal vascular bed (Figure 3D).

The patient was transferred to our clinic with mechanical ventilator support. We accepted the patient to the coronary care unit. The patient's hemodynamic status was unstable, and she was mechanically ventilated. Blood pressure was 80/40 mm Hg and the cardiac pacemaker rhythm rate was 94 beats per minute. The patient was taken to the catheterization laboratory again in our clinic. The first angiographic image showed total occlusion in the proximal RCA and dissection in the ascending aorta (Figure 4). We have observed that contrast agent was preceded to ascending aorta in nearly 2-cm segments. We planned implantation of a stent in the proximal section of the RCA; however, the floppy guidewire did not advance, and we abandoned the PCI. A dissected lumen was seen, and it measured 6 cm in control imaging. Emergency coronary bypass and aortic dissection surgery were planned. It was observed during the operation that the dissection was involving the truncus of the aorta. Ascending aortic grafting and coronary bypass surgery were performed. The patient recovered and was discharged after 10 days.

Discussion

Our presentation has several important clinical points for the interventional cardiologist. We classified the points in this case under 2 topics.

How Can We Distinguish Catheter-Induced Coronary Artery Spasm From a Fixed Coronary Lesion?

Coronary spasm closely resembles a fixed atherosclerotic lesion. The identification of these 2 conditions must be

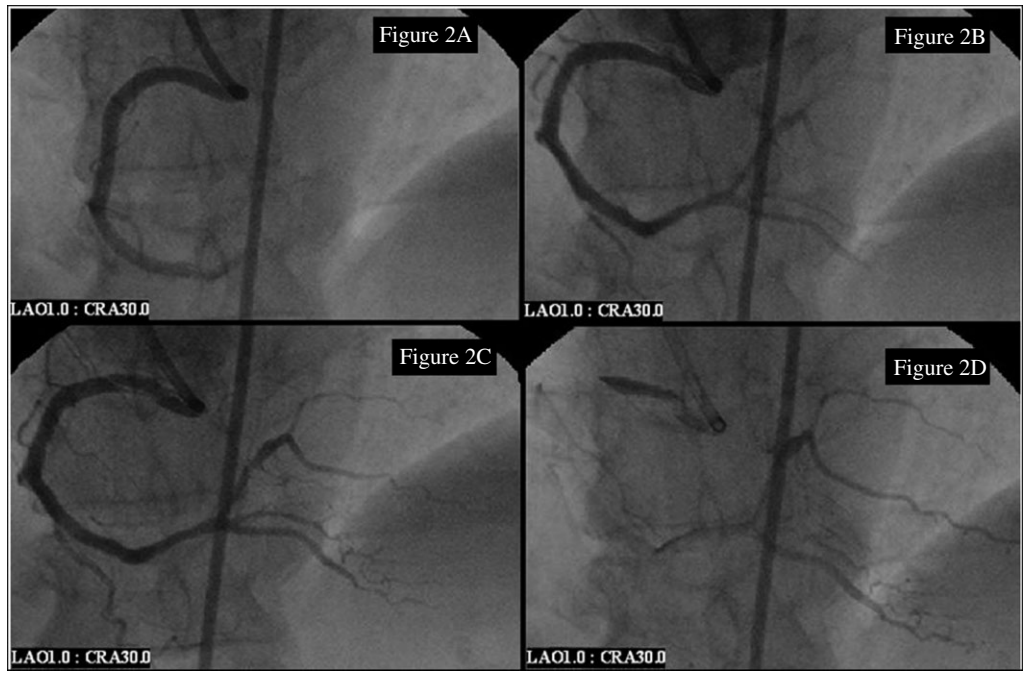


Figure 2. (A–D) Changes in proximal RCA during right cranial imaging. Abbreviations: RCA, right coronary artery.

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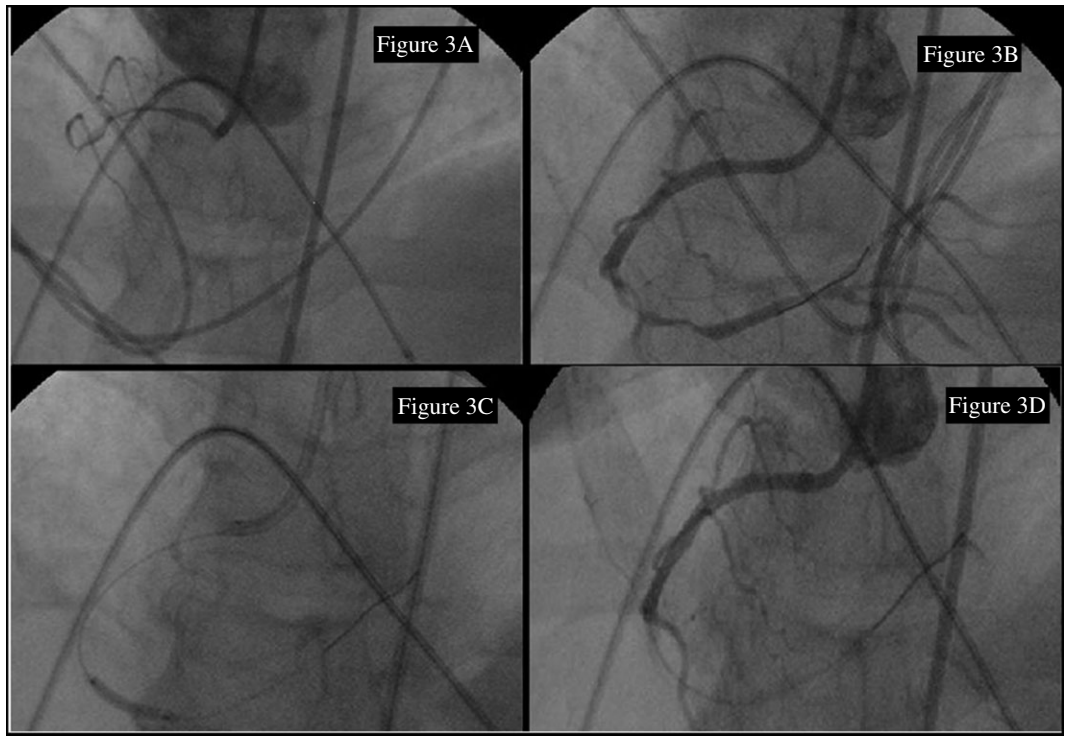


Figure 3. (A–D) From intervention to the total occlusion in the RCA. Abbreviations: RCA, right coronary artery.

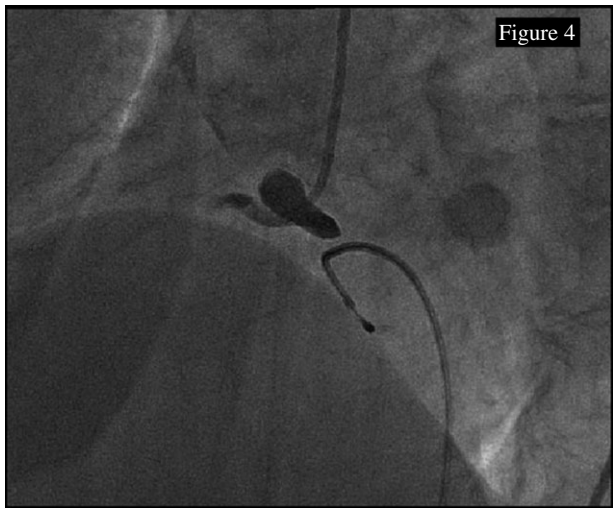


Figure 4. Image of dissection in proximal RCA and ascending aorta. Abbreviations: RCA, right coronary artery.

performed to avoid unnecessary PCI or cardiac surgery. Although coronary spasm is frequently seen in RCA, it may occur in the left main, circumflex, and left descending coronary arteries.[5] Coronary spasm occurs most often in the proximal section of coronary arteries near the catheter as single lesion. It should be noted that distal and multiple spasms may occur in any coronary artery. In most cases, the properties of angiographic lesions may be helpful to identify coronary spasm. The lesion due to coronary spasm is short, concentric, smooth edged, and located in proximal section of the artery. However, in the present case, coronary spasm occurred in the midsection of the RCA and it was seen as a slightly long and eccentric lesion on the angiogram. Identification of this kind of spasm or lesion may be more difficult in clinical practice.

One of most important issues for identification of coronary spasm is administration of nitrate. Half-lives of organic nitrate may vary from 3 to 10 minutes.[6] The time to peak plasma concentration is 1 to 1.5 minutes for intravenous isosorbide dinitrate and 3 minutes for sublingual glycerol trinitrate.[7] It has been demonstrated that nitrate administration by sublingual spray or intracoronary glycerol trinitrate increases equally the diameter of the coronary artery in patients with coronary artery disease.[8] These 2 methods are superior to sublingual glycerol trinitrate. After nitrate administration, the operator must wait for a sufficient time. It has been published that resolution of coronary spasm may proceed for up to 12 minutes after the nitrate administration. In case of doubt, repeated nitrate administration could be given. One of the most appropriate approaches is preprocedural sublingual or intracoronary nitrate administration as routine application in practice for all patients who are undergoing diagnostic coronary angiography. In this present case, coronary spasm or lesion identification was not performed during the first angiography, and this situation led to repeated procedures and subsequent complications.

How Can We Diagnosis and Treat the Coronary Dissection?

The cumulative incidence of catheter-induced dissection with retrograde progression to the aorta has been estimated at approximately 0.008% in diagnostic coronary

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angiography.[4] However, the mortality rate is approximately 50%. Diagnosis and quick intervention are pivotal to decrease this high rate of mortality.

Definition of the type of dissection is also important. The National Heart, Lung, and Blood Institute classifies coronary dissections into 6 types (A–F) based upon their angiographic appearance[9]:

- A: Minor radiolucent areas with little or no persistence of contrast.
- B: Parallel tracts or a double lumen with little or no persistence of contrast.
- C: Contrast outside the coronary lumen with persistence of contrast.
- D: Spiral dissection with filling defects.
- E: Persistent filling defects in lumen.
- F: Total occlusion without distal flow.

In the present case, the initial dissection was type D (Figures 2B–2C). The distal TIMI flow grade (angiographic flow grades based on results of the Thrombolysis In Myocardial Infarction trial) was 3, and the patient had no discomfort. However, severe chest pain had started and hemodynamic status had become impaired, and progressive and advanced type F dissection was seen in control angiography (Figure 3A). It was discussed whether the operator should have inserted 1 or multiple stents into the proximal RCA although distal flow is sufficient. Whereas type A and B dissections are accepted as clinically benign, types C–F have high morbidity and mortality.[10] The most dangerous complication is disappearance of distal flow, as shown in the present case. Because of rapid progression, operators must be more careful, especially in type D and E dissections.

After the hemodynamic impairment, total occlusion was seen. The operator inserted a guidewire into the RCA and administered intracoronary nitrate. Interestingly, the proximal section of the RCA was nearly normal, but there was severe stenosis in the midsection (Figure 3B). The RCA midsection was dilated with a 2.5 × 20-mm balloon, and this dilation caused blockage of distal flow. But progression of the dissection to the midsection of the RCA, instead of coronary spasm, should have been considered initially. It is thought that the operator must have replaced direct stent to stenotic segment without balloon dilation. In these patients, hemodynamic status is unstable due to severe ischemia. In addition, anterograde progression was seen. Therefore, emergency surgery was performed.

This case should call to the attention of the interventional cardiologist these issues: (1) discrimination between coronary spasm and fixed lesion, (2) early diagnosis of coronary dissection, and (3) the possibility of anterograde and retrograde progression of the dissection.

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