Concurrent Dissections of the Ascending Aorta and Right Coronary Artery During Percutaneous Coronary Intervention: A Case Report

CHEN-TUNG HSU, REI-YEUH CHANG, CHENG-KANG CHEN

Iatrogenic aortic dissection caused by coronary angiography or angioplasty is a rare but potentially life-threatening complication. Awareness of the complication and its prompt recognition are important. We describe a 78-year-old woman who underwent coronary angioplasty for acute non-ST elevation myocardial infarction. An ostial dissection of the right coronary artery (RCA) with extension into the entire ascending aorta occurred during the attempt to recanalize an occluded RCA. The literature recommends surgical intervention or stenting of the coronary dissection under these circumstances. However, the patient was managed conservatively. Serial evaluation with noninvasive imaging studies showed resolution of the ascending aortic dissection.

Key words: aortic dissection, coronary dissection, percutaneous coronary intervention

Introduction

Catheter-induced coronary dissection is a well-known complication of angiography and percutaneous coronary intervention (PCI). Extension of dissection into the ascending aorta is a rare, but devastating complication. Twenty-seven percent of Stanford type-A iatrogenic aortic dissections in the International Registry of Aortic Dissections (IRAD) were caused by coronary angiography or interventions. The overall mortality among patients with iatrogenic aortic dissection was not significantly different from that observed for patients with spontaneous aortic dissection (32% vs 35%) (1). However, treatment of iatrogenic dissection of the ascending aorta is challenging and may present a dilemma. We describe a patient who had concurrent dissections of the ascending aorta and right coronary artery (RCA) during an attempt to recanalize an occluded RCA. We also compare our therapeutic strategy and outcome to previously reported cases.

Case Report

A 78-year-old woman was admitted to our hospital with sudden onset of dyspnea for eight hours. She had a long history of type 2 diabetes mellitus, diabetic nephropathy and anemia. She had undergone successful stenting (bare metal stent) of the proximal RCA two years previously at another hospital. Her medications consisted of aspirin 100 mg, trichlormethiazide 2 mg, and insulin 39 units daily. On arrival in the intensive care unit, the patient was intubated and artificially ventilated because of respiratory failure. On physical
examination, her blood pressure was 110/72 mmHg, pulse 116 beats per minute, and temperature 36°C. Diffuse crackles were noted in both lung fields. There was a grade II systolic murmur over the left sternal border and apex. The physical examination showed no other abnormalities. Blood tests were as follows; white blood cells 9180 /uL (normal range: 3500-9900); hemoglobin 6.8 g/dL (normal range: 14-18); platelets 187000 /uL (normal range: 130000-340000); blood urea nitrogen 77.2 mg/dl (normal range: 8-23); creatinine 4.5 mg/dl (normal range 0.7-1.5); creatine kinase (CK) 200 IU/L (normal range 26-174); CK-MB 6.3 IU/L (normal range 0.1-6.3); and troponin I 0.75 ng/ml (normal <0.5). The admission electrocardiogram revealed sinus rhythm, poor R-wave progression, and 1mm ST depression over leads V5, V6, I, aVL, II, III and aVF. Her chest radiograph demonstrated acute pulmonary edema. Nitroglycerin infusion 5 ug/min and furosemide 20 mg intravenous twice daily were administered. Blood transfusion with two units packed red blood cells was given for anemia. Twelve hours later, follow-up cardiac markers of myocardial damage were, CK 592 IU/l, CK-MB 26.1 IU/L, and troponin I 19.01 ng/ml. Her cardiac enzymes peaked 36 hours later with a CK level of 818 IU/L. The next day, a cardiologist was consulted for further management of acute myocardial infarction. The patient was treated with aspirin 100mg daily, clopidogrel 75 mg daily, and subcutaneous enoxaparin 20 mg twice daily for non-ST elevation myocardial infarction. Transthoracic echocardiography showed inferior wall hypokinesis with impaired left ventricular function (ejection fraction 41%), moderate mitral regurgitation and moderate tricuspid regurgitation. After informed consent was obtained, coronary angiography was performed via the right femoral artery, revealing in-stent occlusion of the proximal RCA, with distal collateralization by the left coronary artery (Fig. 1, left panel). Left coronary angiography showed 75% tubular stenosis at the middle of the left anterior descending artery and 60% tubular stenosis at the proximal circumflex artery. Left ventriculography was not performed due to impaired renal function. The option of coronary artery bypass grafting was discussed with the patient and her family, but they refused.

Because we suspected the RCA was the infarct-related artery, PCI to the occluded RCA was attempted first. A 6 French Judkins right 4 guiding catheter (Cordis Corporation, Miami Lakes, Florida, USA) was engaged in the ostium of the RCA but the catheter support and coaxial alignment were suboptimal. The catheter was replaced by a 6 French Amplatz Left 1 guiding catheter (Cordis) which provided reasonable but not perfect back-up force. A 0.014-inch Runthrough wire (Terumo Corporation, Tokyo, Japan) and a 0.014-inch intermediate wire (Guidant Corporation, Santa Clara, California, USA) were used to cross the proximal RCA occlusion. Unfortunately, the lesion could not be crossed even after additional support with a 2.75 × 20 mm Voyager dilation catheter (Abbott Vascular, Santa Clara, California, USA). Although the guiding catheter was deeply engaged, the back-up force was not sufficient. During attempts to advance the wires, the position of the guiding catheter was lost. After the position was reestablished and an injection of contrast medium, concurrent dissections of the ascending aorta and ostial RCA were clearly seen with marked retention of contrast (Fig. 1, right panel). The patient had no specific complaints and remained hemodynamically stable. The PCI procedure was ceased immediately. A follow-up electrocardiogram did not show any dynamic ST-T changes. Emergency computed tomography demonstrated an aortic intramural hematoma (Stanford type A, DeBakey type II) extending 60mm superiorly from the origin of the RCA into the ascending aorta (Fig. 2, upper panels). A transthoracic echocardiogram performed just after
Fig. 1  Left panel: Left anterior oblique view showing total occlusion of the proximal right coronary artery. Right panel: Extensive ascending aorta and ostial RCA dissection during PCI

Fig. 2  Top panels: A computed tomogram immediately after the failed PCI attempt showing an aortic dissection from the right coronary cusp to the ascending aorta with pooling of the contrast medium. Bottom panels (left, T1 weighted; right, T2 weighted): Magnetic resonance imaging 45 days later demonstrating nearly complete healing of the aortic dissection
the dissections revealed no aortic valve regurgitation and no pericardial effusion.

Because the patient refused emergency coronary artery bypass grafting and surgical repair of the aortic dissection, she was managed conservatively without any anticoagulant or antiplatelet agents. The patient was transferred to the intensive care unit where her blood pressure was maintained around 110/70 mmHg with nitroglycerin infusion 5 ug/min and furosemide 20 mg intravenous twice daily. Her heart rate was about 70 beats per minute. We did not prescribe beta-blockers because of acute lung edema. Hemodialysis was performed once after the PCI procedure. Regular hemodialysis was not needed since urine output was adequate and follow-up renal function tests did not show obvious deterioration.

Daily transthoracic echocardiography did not show any new findings. Two days after the event, repeat transesophageal echocardiography revealed an intact ascending aorta without evidence of an intimal flap, pericardial effusion or aortic regurgitation. The patient was discharged uneventfully 15 days later. Thoracic magnetic resonance imaging 45 days after this event demonstrated nearly complete healing with effacement of intramural hematoma (Fig. 2, lower panels). The patient refused elective coronary artery bypass grafting and remained asymptomatic 7 months later on a regimen of clopidogrel 75 mg daily, isosorbide dinitrate 10 mg three times daily, and furosemide 20 mg daily.

**Discussion**

Iatrogenic aortic dissection caused by coronary angiography or angioplasty is a rare but potentially catastrophic event. The incidence of catheter-induced aortic dissection is 0.008-0.02% for diagnostic coronary angiography and 0.06-0.15% for PCI\(^2\)\(^-\)\(^5\). The incidence of iatrogenic aortic dissection is higher (0.19%) in the setting of acute myocardial infarction than in elective procedures\(^2\). With the advent of more complex interventions such as left main stenting, revascularization of chronic total occlusions and mechanical thrombectomy, this catheter-induced complication may become more prevalent.

In most previously reported cases (as in our case), aortic dissections resulted from extension of a proximal coronary dissection, mainly in the RCA. The RCA may be more susceptible to retrograde aortocoronary dissection than the left coronary because (1) the diameter of the left coronary artery is larger than that of the RCA; (2) the angle of the ascending aorta with the left coronary artery is acute, possibly providing a better approach for catheterization; and (3) the periostial wall and sinotubular junction of the left coronary artery are formed by more smooth muscle cells and by a dense matrix of collagen type-I fibers\(^6\).

Predisposing factors for catheter-induced aortic dissection include hypertension, aging, calcification of the aortic root or coronary lesion site, recent myocardial infarction and intra-aortic balloon pump support\(^5\). In our patient, aging and acute myocardial infarction might have been the predisposing factors for aortic dissection. The aging process in medial cystic degeneration of the aorta as well as increased vascular inflammation during acute myocardial infarction might facilitate this complication. In addition, attempts to reopen total occlusions may pose a greater risk of luminal dissection as more aggressive device manipulation is needed. In addition, Dunning et al reported that left Amplatz guides were involved in a disproportionate number of catheter-induced aortocoronary dissections\(^2\). The selection of guiding catheter is a risk-benefit tradeoff between extra support of the guiding catheter and the possibility of coronary artery dissection\(^7\). In our case, the trigger for the aortic dissection might have
been an ostial dissection of the RCA produced by
agegressive manipulation of the left Amplatz guiding
catheter. The retrograde extension of the dissection
toward the ascending aorta was possibly caused by
a subintimal injection of contrast medium. From
the technical viewpoint, an appropriate guiding
catheter, careful manipulation of the catheter, and
optimal coaxial alignment of the catheter as well
as gentle contrast injection are recommended to
prevent iatrogenic dissection.

Therapeutic strategies include surgical
intervention, stenting the entry point of the
coronary dissection, and conservative treatment. Dunning et al have proposed a classification
of iatrogenic aortocoronary dissection to guide
the choice of optimal therapeutic strategy. They reported that seven patients with limited
aortic involvement (Class I and Class II) were
successfully managed with stenting. Extensive
dissection extending from the coronary cusp up
the ascending aorta >40 mm was associated with
the worst prognosis\(^2\). Conservative management
is a reasonable option only in hemodynamically
stable patients with localized aortic dissection.
Sinus of Valsalva dissections that remain localized
during catheterization tend to resolve spontaneously
in the first month\(^3\). Nevertheless, conservative
treatment is unsuccessful in 50% of cases due to
myocardial infarction, progression of retrograde
dissection or death\(^5\). Some reports have shown
that coronary stenting can be a suitable and life-
saving therapeutic option for the patient\(^2,3,5\). If the
entry of the dissection is within a coronary artery
and the retrograde dissection dose not extend more
than 40 mm (class I and class II), the recommended
approach is to stent whenever possible with the
aim of sealing the dissection. However, sealing the
entry of the dissection with coronary stenting may
be inapplicable in 25% of reported patients with
retrograde aortic dissection because of acute severe
aortic regurgitation, hemopericardium, unstable
hemodynamics, intractable chest pain, failure of
the guidewire to cross the occluded lesion (as in our
patient), and left main retrograde dissection\(^5\). These
patients inevitably require surgical management.

The treatment of an extensive aortic dissection
is still controversial. Sixteen cases of class III
catheter-induced aortic dissection have been
reported in detail in the literature (Table 1)\(^2,3,8-19\).
Of these, 56% (n=9) were treated surgically with
four deaths, 31% (n=5) were treated with intra-
coronary stenting, and 13% (n=2) were treated
conservatively. Overall in-hospital mortality rate
of patients with this complication was 25% (n=4).
In the majority of reported cases (75%, n=12), the
RCA was the culprit vessel. Both of the patients
treated conservatively survived but one patient had
a myocardial infarction. Of the patients treated with
coronary stenting, three were treated successfully
by sealing off the aortic dissection, one required
subsequent surgical intervention because of
progressive aortic dissection up to the arch, and
one had a chronic aortic dissection demonstrated
by computed tomography scan at 6 months and was
asymptomatic at 36 months. Coronary stenting may
or may not work, but it is worth trying. However,
a cardiovascular surgeon should be consulted
early. In the case of extensive dissection, surgical
intervention is usually the preferred option. In
addition to optimal blood pressure and heart rate
control, conservative treatment should include
serial imaging studies to monitor progression or
regression of the aortic dissection. Stent deploy-
ment was not done in our patient because of the
wire crossing failure, as well as the stable clinical
condition.

A spontaneous aortic dissection (Stanford
type A or DeBakey type I or II) is considered a
surgical emergency because of the high risk of
threatening complications, such as aortic rupture,
stroke, visceral ischemia and cardiac tamponade.
In one report, the in-hospital mortality was 27%
Successful coronary stenting results in good long-term survival and should be considered of initial management for catheter-induced aortic dissection.

In summary, we describe a patient who had an ostial dissection of the RCA with retrograde extension into the ascending aorta that occurred during an attempt to recanalize an occluded RCA. Sealing the entry of the dissection with coronary stenting appeared inapplicable to our patient. Emergency surgery is often the option under the circumstances we described. However, this case illustrates that a conservative approach in special situations could be another choice in a hemodynamically stable patient with extensive dissection of the ascending aorta.

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RCA = right coronary artery; LCA = left coronary artery

and 56% for surgical and medical therapy, respectively<sup>20</sup>. Although the IRAD reports no differences in overall mortality<sup>1</sup>, there are differences between spontaneous and catheter-induced dissections of the ascending aorta. Significant degeneration of the media is believed to be a prerequisite for the development of spontaneous aortic dissection. On the other hand, only a few cases with extensive dissection were determined to have cystic medial necrosis on histological examination. Dunning described two cases with low grade cystic medial necrosis, which is a common finding with aging<sup>21</sup>. Another important difference is the entry site for the aortic dissection. The entry of a catheter-induced dissection is usually within a coronary artery.
References


冠狀動脈介入治療引發升主動脈剝離：
病例報告及文獻回顧

許振東  張瑞月  陳政康

冠狀動脈介入治療引發主動脈剝離是一種罕見但可能致命的併發症。熟悉此併發症，並能及早發現
和給予適當的處理是非常重要的。我們報告一名78歲的女性，因急性心肌梗塞接受心導管及介入性治
療。當我們試著打通阻塞的右冠狀動脈時，併發右冠狀動脈開口處剝離，並往後延伸至升主動脈。文獻
回顧發現此狀況通常需要外科手術治療，或者冠狀動脈開口處支架植入。然而，此病人接受內科保守治
療後，經一系列的非侵入性影像學檢查，證實升主動脈剝離已癒合。

關鍵詞：主動脈剝離，冠狀動脈剝離，冠狀動脈介入治療