Percutaneous coronary stenting in guide-induced aortocoronary dissection: angiographic and CT findings

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Abstract

We report a case of accidental aortocoronary dissection that occurred during the engagement of a guiding catheter. This resulted in an antegrade dissection into the right coronary artery, and a retrograde extension of the dissection into the Sinus of Valsalva and the ascending aorta. It was successfully treated with a stent deployment at the RCA ostium; this restored ptimal coronary blood flow and there was a complete resolution of the aortic dissection as documented by the follow-up CT scan.

Key words: sinus of Valsalva dissection; aortic dissection; coronary angiography; complications

Introduction

Catheter-induced dissection is well known and uncommon complication of coronary angioplasty, and its natural history is progression to complete coronary occlusion.1 It can be successfully treated with intracoronary stenting.2 Coronary dissection with retrograde extension into the Sinus of Valsalva and the ascending aorta is rare.3-5 The outcome of intracoronary stenting is not known as to whether the dissection will remain stable to the localized areas of the aortic wall or if there will be progression of the dissection.6-7 CT is one of the noninvasive diagnostic tools for evaluating this disease's natural course and the clinical outcome. This case report describes a patient with catheter-induced dissection of the right coronary artery and aortic dissection that was the result of complications from percutaneous intervention. The patient’s condition was successfully treated with stenting, and this was documented by coronary angiography and serial CT scan follow-up.

Case report

A 78-year-old male was admitted to our hospital for further evaluation of chest pain. He had 3 risk factors (diabetes, hypertension, hyperlipidemia). He suffered severe chest pain 4 days ago and visited local hospital. His ECG showed ST-T wave changes in II, III, aVF. Coronary angiography showed total obstruction of mid-RCA with collateral flow from LAD (Fig 1-A) So we decided to treat culprit lesion in mid-RCA. At first we tried to insert the guidewire but failed. By contralateral injection, we could guess the right course of RCA.

After success of guidewire insertion, (Fig 1-B) we inflated 2.5 * 20 mm balloon up to 10 atm 3 times and tried to insert the stent (Cypher, 2.75 * 28mm,
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Fig 1. Coronary angiogram showing total obstruction of mid-RCA (A) and success of guidewire insertion to the distal RCA (B).

Fig 2. Dissection of the proximal RCA (A) with retrograde aortic dissection (B).

Johnson & Johnson, Vm) but failed to insert the stent and guiding catheter, guidewire, and balloon were removed from RCA OS lesion. Suddenly, The patient complained of severe chest pain. A subsequent hand injection of contrast agent demonstrated a proximal dissection of the RCA ostium extending antegrade down to the mid RCA, and the dissection also passed retrograde up to the aortic root(Figure 2-A, 2-B).

After reengaging the jenkins Left 4 guiding catheter, we tried to insert the A soft-tip 0.014" guidewire (Runthrough, Terumo Co, Japan) and it was carefully advanced into the distal RCA to avoid getting into the subintimal tissue. After ballooning the lesion from mid-RCA to proximal- RCA, a stent (Taxus, 2.75*20 mm, Boston-Scientific, MA) was deployed at the mid RCA to prevent propagation of dissection to distal coronary artery and another stent (Taxus, 3.0*32 mm, Boston-Scientific, MA) was deployed at the proximal
RCA lesion. The final stent (Taxus, 3.5*16mm, Boston-Scientific, MA) was deployed at the ostium of the RCA to close the suspected dissection entry site (Fig 3).

There was no residual stenosis in the RCA and no further extravasations of contrast agent on the final angiogram (Figure 3B). The patient was stabilized at the cath lab with complete resolution of his ST segment elevation.

When he was transferred to intensive care unit, V-fib was detected and DC cardioversion was done.

An urgent thoracic CT after angioplasty was performed to decide if there was a need for surgical intervention. The CT scan revealed an aortic dissection with a considerable amount of subintimal hemorrhage at the ascending aorta (Figure 4A and B).

The patient was observed for 4 days in the coronary care unit, and he showed no signs of chest pain. The
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12 lead ECG showed no changes compared with the baseline ECG. The peak serum CK-MB was 14.8 ng/ml (upper limit <6.0 ng/ml) and peak serum troponin T was 2.5 ng/ml (upper limit <0.1 ng/ml). Aspirin 100 mg, clopidogrel 75 mg and cilostazol 200 mg were continued without interruption. The patient was discharged 7 days after admission in a good condition.

Discussion

Grunzig revolutionised the management of coronary artery disease with first successful PTCA. Interventional cardiologist are using this technique in increasingly difficult cases, thus increasing chances of complications such as coronary dissection, coronary perforation, acute coronary syndrome and dysaarrhythmias.

Dissection of the ostium of the RCA involving the right sinus of Valsalva is potentially a life-threatening condition; its management depends on patient’s conditions, progression of aortic dissection, and underlying coronary anatomy. While some authors maintain that the best option is outright surgical repair, others contend that medical management should be the initial option if the patient is stable and the dissection limited to the sinus of Valsalva. However, even when the initial clinical presentation is apparently benign, progression of the dissection into the ascending aorta can suddenly develop; not surprisingly, all cases managed conservatively had a fatal outcome.

Various complications during percutaneous coronary revascularization have been reported in the literature, however, extensive dissection of coronary arteries with retrograde dissection to the CSV during PTCA, as occurred in our patients, has rarely been reported.

The etiologies of coronary dissection complicating PTCA have been emphasized due to the use of rigid wires, forceful manipulations of guiding catheters and balloon catheters, and vigorous contrast medium injections. However, the exact mechanism responsible for the propagation of dissection and the occurrence of aortic dissection remains to be established. We think that two mechanisms could be responsible for this distinctive complication in our patients. (1) Contrast injections caused an already existing dissection to progress. This mechanism, derived from the angiographic observations in our patients also was hypothesized by Perez-Castellano and colleagues and other authors. (2) The shearing forces of blood flow during systole and diastole could also probably explain the antegrade and retrograde propagation of the dissection. This is just like the mechanism that causes an aortic dissection in a hypertensive patient.

The entry ports that were created by mechanical trauma and/or forceful injection of contrast medium into the subintimal space had already been exposed to the aortic bloodstream, which, in turn, contributed to the subsequent extension of the dissection. This reasonable explanation further supports our second hypothesis. These two mechanisms support the theory, which had been suggested by Perez-Castellano et al., that dissection of the right CSV always results from a retrograde extension of a dissection of the RCA and could be extrapolated to explain the retrograde dissection of the left CSV. Catheter induced aortocoronary dissection has been treated with conservative treatment, stenting or surgery. Catheter induced aortocoronary dissection can be treated with a stent if the inlet of the dissection is well defined and the stent is sufficient to cover the dissection flap. We performed stenting to seal the inlet of the dissection by quickly implanting the larger sized coronary stent to stop the further progression of aortic dissection, and we finished the whole procedure in a usual manner. In our case, complete sealing of entry site was confirmed by follow-up coronary angiogram with no more contrast leakage and anticoagulation was not reversed.
In conclusion, stenting the entry site of dissection should be the first strategy in catheter-induced aortocoronary dissection, and a serial CT scan was helpful in our case to observe the results.

References