 Unexpected retraction of an implanted coronary stent

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Introduction

The utilization of coronary stent has broken new ground in percutaneous coronary intervention (PCI); however, it has also brought new complications to cardiology, like frequent stent restenosis and stent thrombosis and, rarely, stent fracture and stent dislodgement (1-5). These complications can lead to myocardial infarction, emergency operation, peripheral and cerebral embolism, and even death (2-8). Retrieving the unplaced stent by pulling away the balloon through a device, like a snare catheter, is a rare case in the literature, but the retraction of a placed intracoronary stent through a jailed guidewire has not, to our knowledge, been presented in the literature yet (5).

Case Report

A 69-year-old female patient with diabetes and end-stage renal disease was hospitalized with a diagnosis of non-ST elevation myocardial infarction (NSTEMI). The ECG showed lateral ST segment depressions. The GRACE and CRUSADE risk scores of the patient were 133 and 75, respectively. She was planned for early cardiac catheterization after a nephrology consultation. Coronary angiography revealed 50% diffuse stenosis at the mid- and distal left anterior descending artery (Videos 1, 2), 90% severe calcified eccentric stenosis at the mid-circumflex artery (Cx) (Video 3), and 30% stenosis at the mid- and distal right coronary artery (Video 4). Percutaneous coronary intervention (PCI) of the Cx artery was planned after a consultation by cardiovascular surgery. Cx was engaged with a 6-Fr guiding catheter (JL4, Cordis, Miami Lakes, FL, USA), and predilatation was performed with a Sprinter 1.25x15-mm balloon (Medtronic, Minneapolis, MN, USA) (Video 5). We tried to pass the lesion with a 2.5x18-mm zotarolimus-eluting coronary stent (Endeavor, Medtronic, USA). But, owing to the tortuosity at the proximal segment of the Cx artery, we were not able to deliver the stent to the lesion. Therefore, a second guidewire was delivered distal to the lesion in the branch, and it was anchored. A repeat balloon angioplasty was performed with a 2.0 x 15-mm balloon, and after that, a stent was delivered to the lesion and implanted successfully at 8 atm pressure (Videos 6, 7).

After implantation of the stent, we tried to pull back the guidewire, which was jailed between the stent and vessel wall. But, this attempt was unsuccessful. Therefore, we increased the force for pulling both guidewires; consequently, both guidewires were pulled back. But, unexpectedly, we saw that the implanted stent was also removed. The stent was highly deformed, and its structure was damaged (Fig. 1). The patient’s hemodynamic status was stable, and she had no complaint of angina. Coronary angiography showed TIMI grade 3 flow with no clear dissection in either the Cx or left main artery. We again tried to deliver a new stent, but our attempt was unsuccessful. There was no lesion blocking the coronary flow in the Cx, and the patient had end-stage renal failure; therefore, we decided to end the procedure (Video 8). She was discharged with anti-anginal medications and dual antiplatelet therapy. At an outpatient visit 1 month after discharge, she was comfortable with the medical therapy.

Discussion

We proposed two mechanisms underlying this complication. According to the first scenario, the guidewire that was trapped between stent struts and a tight calcific plaque might behave like a hook and thereby cause retrieval of the stent with the operator’s forceful maneuver while pulling back the guidewire. A second possibility is that the distal portions of the guidewires might be wrapped around each other, and application of force from the outside to pull back might have resulted in stent retrieval.

This kind of complication can be very serious, resulting in arterial dissection, intracoronary or extracoronary embolism, and even sudden death (2, 6, 8). Therefore, we suggest that particular attention should be paid to the removal of second guidewire after stent delivery to the tortuous and calcific lesions and just before deployment of the stent. If there is a need to leave a second guidewire in place for side branch protection, we suggest inflating the stent with low pressure (6 atm) first, and after removal of the side branch wire, inflation with higher pressures can be achieved. If there is still failure of removal of the guidewire, a low-pressure chronic total occlusion balloon can be advanced to the site and inflated with minimal pressure.

Conclusion

A second guidewire is generally used for supporting or protecting the branch in complex PCI. There is always a possibility of trapping the second guidewire, which can cause serious complications, although it occurs rarely. Therefore, the operator should pay attention to protect trapping of the second guidewire.

Video 1. Coronary angiogram showing lesion at proximal and mid-left descending coronary artery
Video 2. Coronary angiogram showing lesion at mid- and distal left descending coronary artery
Video 3. Coronary angiogram showing lesion at circumflex artery
Video 4. Coronary angiogram showing lesion at right coronary artery
Video 5. Coronary angiogram showing inflation of 1.25 x 15-mm balloon
Video 6. Coronary angiogram showing inflation of 2.0 x 15-mm balloon
Video 7. Coronary angiogram showing stent placement
Video 8. Coronary angiogram after retrieval of stent
A clinical dilemma about a new oral anticoagulant treatment

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Introduction

Atrial fibrillation (AF) is the most common chronic cardiac arrhythmia (1, 2). Major mortality and morbidity are associated with stroke and systemic embolism in patients with AF (3). The CHA2DS2-VASc is a clinical score for estimating the risk of stroke in patients with non-valvular AF and is used to determine whether anticoagulation therapy treatment is required or not (2-4). The numerous limitations of the clinical usage of warfarin have led clinicians to search for alternative agents. New oral anticoagulants (NOACs), such as dabigatran, appear to be preferable in these patients (5, 6). Herein, we present a patient with acute ischemic stroke (AIS) occurring under dabigatran treatment, causing fainting, which resulted in a traumatic large lower leg hematoma.

Case Report

An 82-year-old lethargic female patient was admitted to our emergency department with complaint of sudden loss of consciousness. On physical examination, a traumatic large hematoma (21x16 cm) was noticed on her right lower leg. On neurological examination, motor aphasia and right hemiplegia were observed. Ten months ago, she had been diagnosed with a transient ischemic attack, persistent AF, and hypertension. Based on the European Society of Cardiology (ESC) Committee Guidelines (2), she had been considered to be in a high-risk group (CHA2DS2-VASc score: 6 points), and 110 mg oral dabigatran (b.i.d.) had been initiated as an anticoagulant. Brain computerized tomography showed hypoattenuation and sulcal effacement in the left middle cerebral artery distribution (arrows).

Figure 1. A, B. Axial NECT (non-enhanced computerized tomography) images show hypoattenuation and sulcal effacement in the left middle cerebral artery distribution (arrows)

Figure 2. A photograph of the large hematoma after linear incision for drainage

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Available Online Date: 23.10.2014

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DOI:10.5152/akd.2014.5609