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Case Report Open Access

Inappropriate Stent Placement Resulting in a Coronary Slow-Flow: Case Report

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Abstract

A case of primary percutaneous coronary intervention (PCI) is shown in the elderly patients with an ST-segment myocardial infarction. Stent deployment following the extensive thrombus aspiration resulted in a slow-flow phenomenon. Optical coherence tomography imaging showed a huge thin-capped fibro-atheroma encroaching the landing zone for stent placement. The author discusses possible factors involved in the development of the slow-flow.

Keywords: Primary PCI; OCT intravascular imaging; slow-flow; Thin-capped fibro-atheroma; Inappropriate landing zone

Introduction

Primary percutaneous coronary intervention (PCI) is the standard care for patients with an ST-segment elevation myocardial infarction (STEMI) [1]. However, there are some patients who seem not to benefit fully from prompt restoration of antegrade flow, as they fail to show resolution of signs of ischemia and improvement in perfusion abnormalities. These patients present an angiographic phenomenon characterized by evidence of slow-flow (SF) in the affected vessel (Thrombolysis in Myocardial Infarction [TIMI] flow grade ≤2) and lack of contrast uptake "blush" despite the absence of mechanical obstructions. The prevalence of SF has been reported as 0.2-30% in various studies [2]. SF has a multifactorial pathogenesis including distal embolization, ischemia-reperfusion injury, and individual predisposition. It is possible that the importance of each pathogenetic component is different in various patients, thus explaining the occurrence of SF despite the use of mechanical thrombus aspiration. A series of consistent data has clearly shown that SF has a strong negative impact on outcome, negating the potential benefit of primary PCI [3]. We report, therefore, a case of a patient with STEMI who developed SF despite the extensive thrombus aspiration. Using the optical coherence tomography (OCT), we proposed that SF might have developed as a consequence of inappropriate stent positioning.

Case Report

A 78-year-old lady with hypertension presented to our hospital with severe, persistent chest pain. On admission, her respiratory rate was 12/min, oxygen saturation 93%, heart rate 70/min, blood pressure 125/80 mm Hg, with basal crackles. In the electrocardiogram (ECG), there were marked ST-segment elevations in the leads II, III, aVF, and V6. She was immediately taken to the catheterization laboratory. The emergent coronary angiography revealed a tight (~95%), long lesion with a large filling defect in the middle right coronary artery (RCA); yet distal blood flow was rather preserved (Figure 1a). Left coronary system was unremarkable. At that point, OCT imaging was performed using the C7 Dragonfly catheter and the IlumienTM system (St. Jude Medical Europe, Inc., Zaventem, Belgium).

The imaging discovered a massive fibro-lipidic plaque with a large lipid pool and a thin fibrous cap (TCFA), extensive intimal dissection, and mixed thrombus with a long tail (Figure 2a). Furthermore, there was another, huge TCFA ~15 mm proximal to the culprit lesion (Figure 2b). Consequently, the primary PCI was carried out under protection of the unfractionated heparin. Initially, the manual aspiration by means of the ExportR 6F catheter (Medtronic Vascular, Santa Rosa, Ca, USA) yielded large pieces of red thrombus. Two bare-metal stents Multi-link Vision (Abbott Vascular, Santa Clara, CA, USA), 3.0×18 mm distally and 3.0×15 mm proximally, were eventually implanted at 18 Atm and stent overlap was post-dilated at 20 Atm. Repeat angiography showed nicely deployed stents though the peripheral blood blood had slowed down to TIMI grade 2 (Figure 1b) immediately after the implantation of the proximal stent (Figure 1c). Repeat OCT imaging revealed an appropriate expansion of both stents (Figure 3a); however, a few struts of the proximal stent were wedged into the detached TCFA that remained partly uncovered (Figures 3 b,c). As the patient was doing well and the ECG hadn't changed, the PCI was brought to an end without further adjustment. The cardiac troponin I raised to a peak of 20.662 ng/ml (upper limit of normal 0.01 ng/ml) on the same day. The further hospital course was unremarkable and the patient was transferred to the peripheral hospital on the third day, receiving aspirin, clopidogrel, β-blocker, ACE inhibitor, and statin.

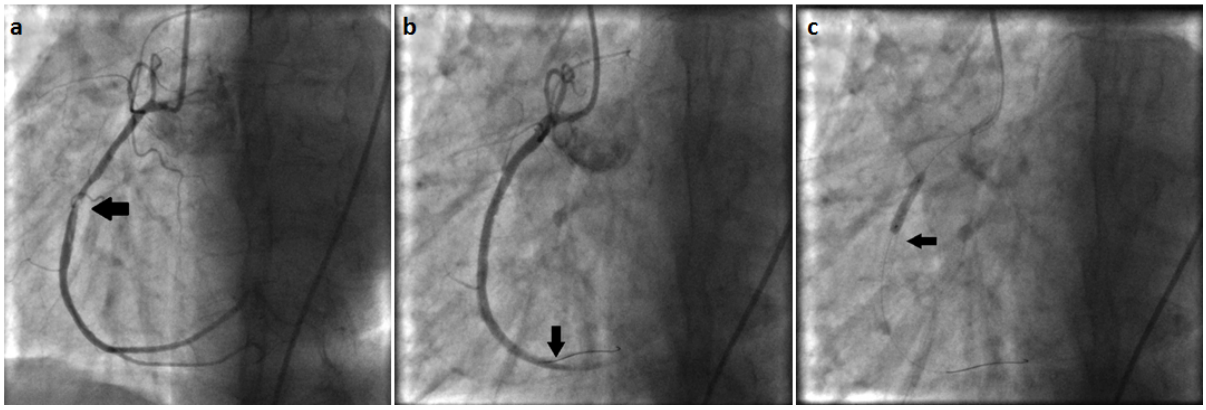

Figure 1: a) Angiogram of the right coronary artery (RCA) initially. Note tight stenosis (black arrow) with a haziness representing intraluminal thrombus, b) Angiogram of the RCA after stenting. Note the slow-flow phenomenon (black arrow), c) Shown is the placement of the second stent in the upper RCA. Note the shadow of the already deployed distal stent (black arrow).

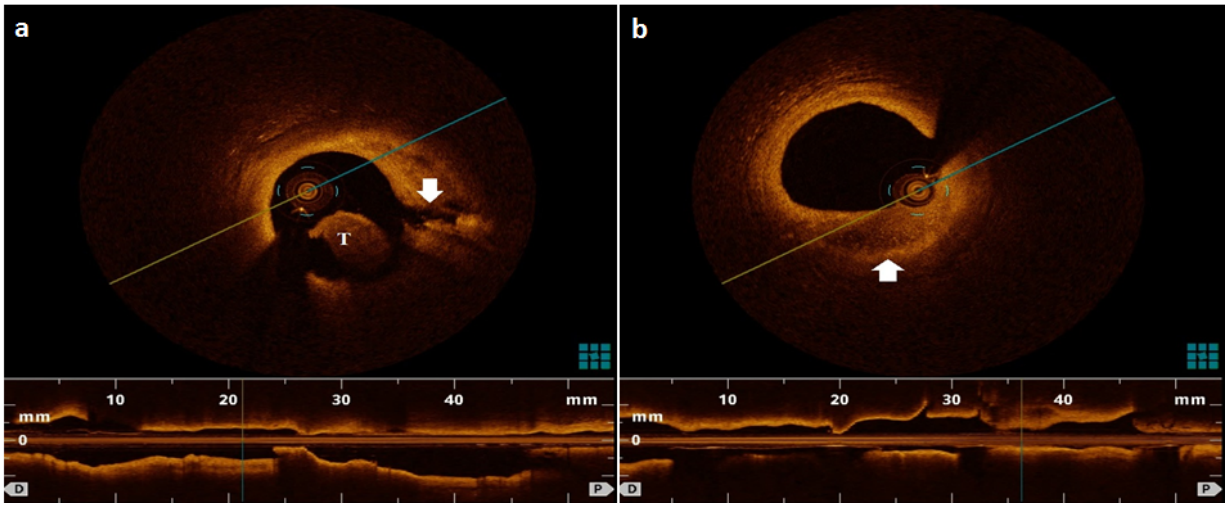

Figure 2: a) OCT image of the culprit lesion beyond the tightest narrowing. Note the intimal dissection (white arrow); thrombus (T) is mostly intraluminal, b) OCT image of the right coronary artery proximal to the culprit lesion. Shown is the thin-capped fibro-atheroma (white arrow); the thinnest part of the fibro cap I seen next to the OCT catheter.

Discussion

We report the case of the elderly patient who had suffered from STEMI caused by the thrombotic obstruction of the RCA. Despite the extensive manual thrombus aspiration, the primary PCI resulted in the SF decreasing the benefit of the achieved reperfusion. The OCT imaging revealed the struts of the proximal stent to be wedged into the detached TCFA.

There has been a growing wealth of scientific literature pointing at the worrying influence of the fibrolipidic plaques, particularly TCFAs, on PCI outcomes. The presence of TCFA by itself has been associated with SF, microvascular obstruction, and peri-procedural myocardial infarction in patients who had undergone elective or urgent PCI [4,5]. Moreover, the volume of the fibro-fatty plaque over the entire length of stenosis, determined by virtual histology intravascular ultrasound imaging, was an independent predictor of SF during primary PCI (beta=0.359, p=0.006) [6]. Tanaka et al. [7] proved that TCFAs, demonstrated by OCT, were more frequently observed in non-STEMI patients who developed PCI-related SF (50% vs. 16% in no-SF patients, p=0.005); furthermore, a multivariable logistic regression analysis revealed that lipid arch wider than 90o was an independent predictor of SF (odds ratio 1.018, p=0.01).

It has been widely known that interventional cardiologists prefer to position stents' edges into normal or at least "less diseases" vessel references. One of the advantages of OCT guidance is that it can help precisely identify the optimal segment for stent placement – the so-called stent landing zone [8]. Recently reported clinical cases have proven pre-procedural OCT's unique ability to avoid late stent failure, revealing at the landing zone the presence of inflamed, lipid-rich core plaques (LCP), undetected by angiography and causing local restenosis [9]. TCFAs at proximal stent edges were also predictors of uncovered

struts [10]. Finally, Hanson et al. [11] demonstrated using near-infrared spectroscopy that LCPs extended beyond angiographic target lesions in nearly 20% of cases. Disturbingly, the failure to cover the longitudinal extent of lesions, particularly if the uncovered lesion segment contained LCP, was associated with increased rate of stent thrombosis and restenosis.

Large thrombus burden has been demonstrated as an independent predictor of major adverse events and stent thrombosis in culprit arteries of patients treated for STEMI [12]. Distal embolization has been repeatedly shown during primary PCI, particularly after stent deployment [13]. Routine manual thrombectomy, as compared with PCI alone, was not able to reduce the composite of clinical adverse events (6.9% vs. 7.0%, p=0.86) in the recent TOTAL trial [14]. Moreover, manual aspiration didn't increase OCT-determined flow area in the TROFI study [15]. One of possible explanations for the aspiration failure might be the inefficiency of the current manual device as appreciated in the study of Imola and co-workers [16,17].

Taken together, the scientific data and the presented case altogether point at the deleterious influence of the inappropriate landing zone for stenting on the development of SF in primary PCI. Certainly, contribution of distal embolization must be taken into account. We argue, however, that SF in our case appeared after placing the second, proximal stent which was possibly trying to avoid the tightest part of the lesion (Figure 1c). The largest amount of the thrombus was seen beyond the stenosis as a long tail (Figure 2a). Co-registration of OCT pullback with X-ray angiogram in our case would definitely prove useful to explain in more detail the cause of SF.

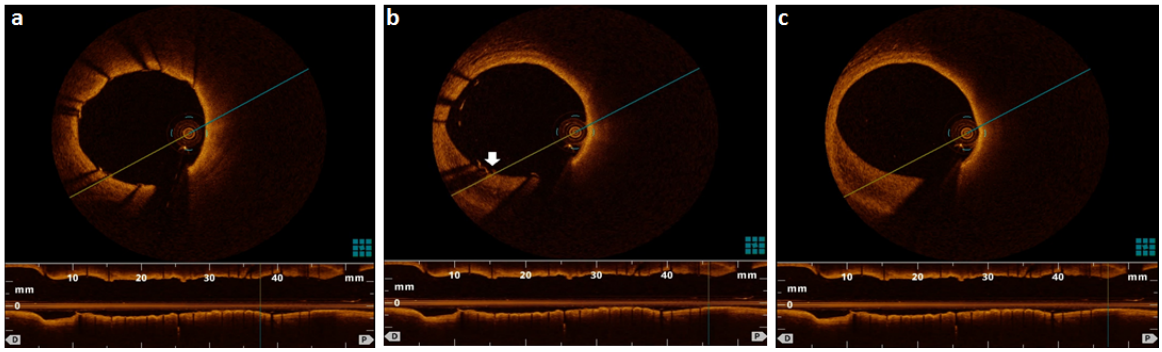

Figure 3: a) OCT image of the right coronary artery after stenting. Note well expanded and apposed stent struts, b) OCT image of the proximal stent edge. Note the wedged stent struts (white arrow) into the thin-capped fibro-atheroma; there are some malapposed struts at 9 o'clock, c) OCT image of the vessel segment adjacent to the proximal stent edge. Note the thin-capped fibro-atheroma at 7 o'clock. Stent struts are no more visible.

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