

CASE REPORT

An Undersized Stent Does Not Forgive

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Abstract

Stent thrombosis is a rare complication of percutaneous coronary intervention (PCI) with potential devastating results. Stent undersizing and malapposition are major risk factors of stent thrombosis and have been strongly associated with early stent thrombosis. However, despite the continuous progress of interventional cardiology, there is no consensus regarding optimal management of this medical emergency. Herein, we present a case of subacute stent thrombosis that manifested as a severe inferior ST-elevation myocardial infarction four days after an initial PCI of the right coronary artery. A stepwise treatment approach was followed aiming initially to achieve flow TIMI III without stenting during primary PCI. During a revision procedure, stent undersizing and malapposition were identified as underlying mechanisms of stent thrombosis using intravascular imaging guidance and treated successfully with corrective PCI. Further evidence is needed to refine the optimal treatment strategy in the setting of stent thrombosis. *Rhythmos* 2019;14(2):31-34.

Key words: drug-eluting stent; stent undersizing; stent malapposition; stent thrombosis

Abbreviations: DES: drug-eluting stents; OCT: optical coherence tomography; PCI: percutaneous coronary intervention; RCA: right coronary artery; STEMI: ST-elevation myocardial infarction

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Introduction

In the era of newer generation drug-eluted stents (DES) and more potent antiplatelet therapy, stent thrombosis remains a major concern in the field of interventional cardiology. Although stent thrombosis is a rare complication of percutaneous coronary intervention (PCI),¹ it is associated with high rates of morbidity and mortality.² Risk factors are recognized to be patient-related, lesion-related, procedure-related, device-related and pharmacotherapy-related. Cumulative evidence suggests that coronary stent undersizing and malapposition are established predictors of stent thrombosis³ and prominent underlying mechanisms regarding early stent thrombosis.⁴ Management of stent thrombosis is challenging for interventional cardiologists as scarce data

are available regarding the optimal treatment strategies in the various clinical scenarios of such a life-threatening complication.⁵

We herein report a case of subacute stent thrombosis occurring 4 days after the initial PCI that apparently was related to stent undersizing and malapposition.

Case presentation

A 73-year-old male presented with severe chest pain, diaphoresis and nausea to our emergency department 2 hours after symptom onset. The electrocardiogram revealed an inferior ST-elevation myocardial infarction (STEMI) with complete atrioventricular block. Four days earlier he had undergone a scheduled coronary angiogram at another institution due to new-onset effort angina. A single vessel coronary artery disease was diagnosed, followed by PCI of the right coronary artery (RCA). No report of the procedure was available at the time of admission in our hospital. The patient had a past medical history of diabetes mellitus, arterial hypertension, chronic obstructive pulmonary disease and obesity. He was on dual antiplatelet therapy with aspirin and clopidogrel, ramipril, β -blocker, atorvastatin, metformin and pantoprazole. Good compliance with the prescribed treatment was reported. On admission the patient was hemodynamically unstable (blood pressure: 80/50 mmHg, heart rate: 35 bpm). The echocardiographic assessment showed a left ventricular ejection fraction of 40% with akinesis of inferior and lateral walls. His initial laboratory results revealed mildly elevated high-sensitivity troponin (30 pg/ml), elevated creatinine (2.1 mg/dl) and hyperglycemia.

He was immediately transferred to the catheterization laboratory for emergency coronary angiography intending to perform primary PCI. The right femoral approach was selected due to cardiogenic shock with complete atrioventricular block. A temporary pacemaker lead was initially inserted from the right femoral vein in order to pace at 80 bpm and improve the hemodynamic condition. Coronary angiography showed no significant lesion in the left coronary artery (Fig. 1A), while there was an acute thrombotic occlusion at the first segment of the RCA, within the previously stented area with two stents extending from the proximal to mid-segment (Fig. 1B). Heavy thrombus burden and TIMI flow 0 were present. Considering the lack of information regarding the stent dimensions and types used in the previous PCI, a decision was made to perform primary PCI without stenting if at all possible. After crossing the RCA occlusion with a conventional guidewire without difficulty (Fig. 1C), thrombus aspiration was performed with restoration of TIMI II flow after two runs (Fig. 1D). To further improve flow, a balloon inflation with a 3x25mm balloon was

performed in the stented area at the mid-RCA (Fig. 1E). Finally, TIMI III flow was restored, despite the significant remaining thrombotic burden in the stented area (Fig. 1F). The vessel environment was judged not suitable for stent implantation, while the patient had already significant clinical improvement with resolution of ST segment elevation and restoration of sinus rhythm. He was transferred to the coronary care unit for monitoring. Antithrombotic treatment after PCI included aspirin, ticagrelor, enoxaparin for 5 days and tirofiban for 48 hours.

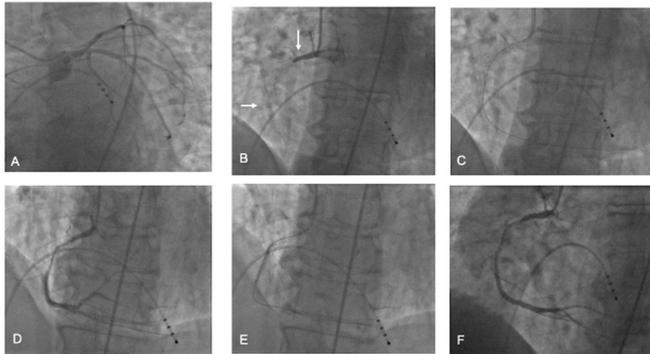


Figure 1. Emergency coronary angiography and primary PCI. **A.** Left coronary artery without significant lesion, **B.** Dominant RCA occluded at the 1st segment, with a stented zone (with 2 consecutive stents) extending from the proximal to mid-segment (arrows show the beginning and the end of the stented zone). Heavy thrombus burden and flow TIMI 0 are noted (without retrograde filling of the distal RCA), **C.** Crossing the occlusion with a conventional guidewire, **D.** Thrombus aspiration with TIMI II flow restoration after the second run, **E.** Balloon inflation with a 3x25 balloon in the stented area at the mid-RCA (at 6 atmospheres for 15 sec), **F.** Final result: TIMI III flow was restored.

A new coronary angiography to image stents with optical coherence tomography (OCT) was scheduled in 6 days. The patient remained symptom free and without complications until then. Meanwhile the previous PCI report and video loops became available and were reviewed. The procedure had been performed through a narrow and tortuous high take-off right radial artery, with great technical difficulty (Fig. 2A). Left coronary artery did not have any significant lesion (Fig. 2B). RCA had a long significant atheroma, extending from the proximal to the mid-segment where a severe stenosis (80%) was noted (Fig. 2C). The lesion was directly stented with a 3x38mm DES (Fig. 2D). A second 3.5x12mm DES was added proximally (with a minimum overlap zone) to cover residual atheroma at the proximal segment and a quite good angiographic result was obtained (Fig. 2E).

Repeat coronary angiography with intracoronary imaging via optical coherence tomography (OCT) was performed 6 days later. The RCA was angiographically improved, free of visible thrombus, with TIMI III flow (Fig. 3).

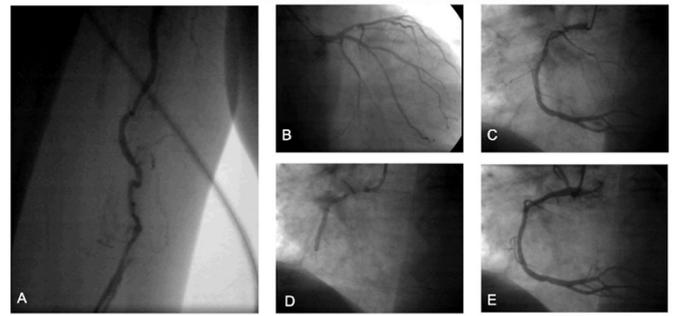


Figure 2. Initial PCI **A.** The procedure had been performed through a high take-off, narrow and tortuous right radial artery, **B.** Left coronary artery without any significant lesion, **C.** Right coronary artery with a long significant atheroma, extending from the proximal to the mid-segment where a severe stenosis (80%) was noted, **D.** Direct stenting of the lesion with a 3x38mm DES, **E.** A second 3.5x12mm DES was added proximally (with a minimum overlap zone) to cover residual atheroma at the proximal segment with a good angiographic result as shown.

OCT examination was chosen in order to understand the subacute stent thrombosis mechanism. The distal reference diameter at mid-RCA was measured around 3.5 mm (Fig. 3A). The distal 3x38 mm stent was mostly well-apposed in mid RCA (Fig. 3B), with limited areas of underexpansion and malapposition (within-stent mean diameter around 2.5mm) (Fig. 3C). At the stent overlap zone (at the end of the proximal RCA segment) the mean diameter was 2.58 mm, while the overlapping stents had nominal diameters of 3 mm & 3.5 mm respectively (Fig. 3D). The proximal part of the proximal RCA stent was significantly undersized and malapposed: the obtained in-stent mean diameter was around 3.3-3.5mm for a reference vessel diameter of ≥ 4.5 mm (Fig. 3E, 3F).

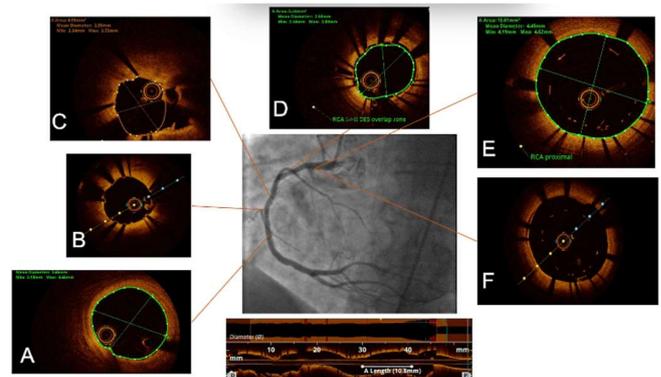


Figure 3. Revision coronary angiogram under OCT-guidance **A.** Distal reference diameter at mid-RCA was measured around 3.5mm, **B.** The 3x38mm stent was mostly well apposed in mid RCA, **C.** Limited areas of underexpansion and malapposition (with in-stent mean diameter around 2.5mm), **D.** At the stent overlap zone the mean diameter was 2.58mm, while the overlapping stents had nominal diameters of 3mm & 3.5mm, **E, F.** The proximal part of the proximal RCA stent was significantly undersized and malapposed: obtained in-stent mean diameter of 3.3-3.5mm for a reference vessel diameter of ≥ 4.5 mm.

Obviously, stent undersizing and malapposition was the most probable mechanism for the subacute stent thrombosis. Before proceeding to corrective PCI, we verified the expansion limits of the DES previously used. The 3x38mm DES extending from the proximal to mid-RCA could be expanded up to 3.85mm and the additional 3.5x12mm DES in the proximal RCA could be expanded up to 4.75mm. With the aim to adapt DES expansion to the vessel's reference diameters and correct undersizing and malapposition we proceeded to PCI. A 3.5x30mm non-compliant balloon was dilated in the stented area distally and then more proximally at 18 Atm (Fig. 4A, 4B). A 4.5x21mm non-compliant balloon was positioned to extend from the proximal DES entry point, through the DES overlap zone and into the proximal part of the more distal DES and was inflated at 14 Atm (Fig. 4C). Considering the 3x38mm stent expansion limit (3.85mm) this balloon was oversized, but it was mostly important to correct undersizing and malapposition of the proximal 3.5x12mm DES (with an expansion limit at 4.75mm) and approach the reference vessel diameter which was at least 4.5mm for the proximal RCA. A good final angiographic result was obtained (Fig. 4D).

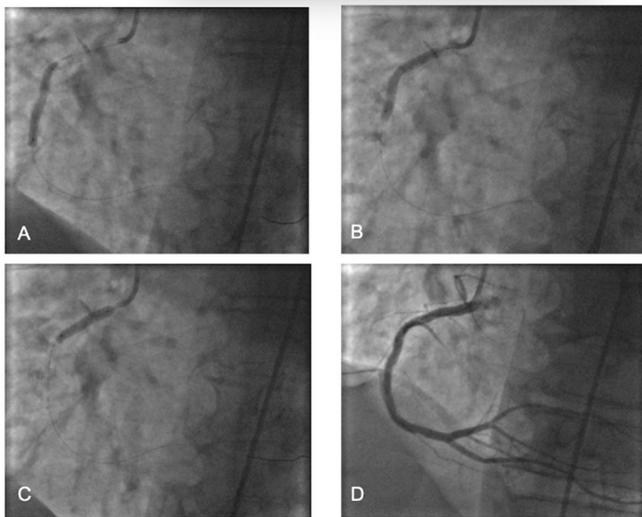


Figure 4. Revision PCI under OCT-guidance **A, B.** Dilation of a 3.5x30mm non-compliant balloon in the stented area distally and then more proximally (18 Atm), **C.** Dilation of a 4.5x21mm non-compliant balloon from the proximal DES entry point, through the DES overlap zone and into the proximal part of the more distal DES (14 Atm), **D.** A satisfactory final angiographic result was obtained.

A final control with OCT revealed good stent expansion and apposition throughout the stented mid-RCA (Fig. 5A, 5B), while the stent overlap zone was now well-expanded with mean diameter around 3.5mm (Fig. 5C). No signs of stent structural disruption at the proximal 8-10mm of the 3x38mm DES were noted after the final post-dilatation with the 4.5x21mm non-compliant balloon. This

final post-dilatation led to improved expansion of the proximal RCA DES, obtaining mostly good apposition with mean stent diameter around 4.5mm at the previous area of undersizing and malapposition (Fig. 5D). At the initial 2mm of the proximal DES minimal malapposition persisted since the reference diameter there was around 4.8mm (slightly beyond the maximum expansion limit of the DES) (Fig. 5E).

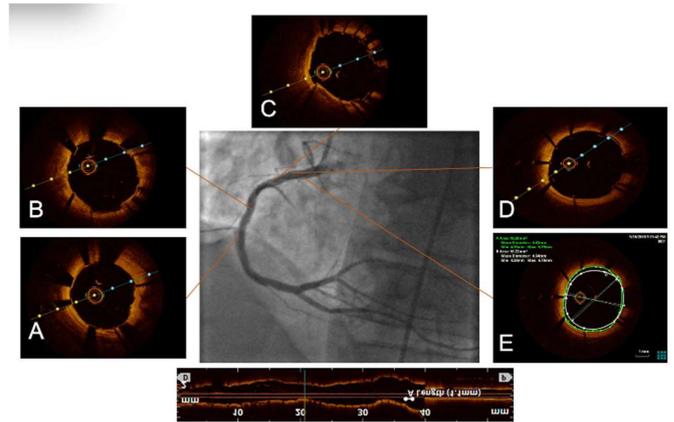


Figure 5. Final control with OCT. **A, B.** Good stent expansion and apposition throughout the stented mid-RCA, **C.** Stent overlap zone well-expanded with mean diameter around 3.5 mm. No signs of stent structural disruption at the proximal 8-10mm of the 3x38mm DES after the final post-dilatation with the 4.5x21mm non-compliant balloon, **D.** Improved expansion of the proximal DES, obtaining mostly good apposition with mean stent diameter ~4.5mm at the previous area of undersizing and malapposition, **E.** At the initial 2mm of the proximal DES minimal malapposition persisted since the reference diameter in this segment was ~4.8mm (slightly beyond the maximum expansion limit of the DES).

The overall result however was considered satisfactory and it was decided not to further overstretch the proximal DES with a post-dilatation with a non-compliant balloon of 5mm diameter. The patient was discharged without complications the following day. There was no adverse event during the initial follow-up at 6 months.

Discussion

We described a case of subacute thrombosis related to undersized and malapposed DES manifesting as an acute inferior STEMI (despite uninterrupted dual antiplatelet therapy) that was successfully treated initially by primary PCI with thrombus aspiration and lesion dilation to achieve TIMI III flow, then with intensive antithrombotic therapy for a few days to obtain residual thrombus dissolution and finally by performing a revision OCT-guided PCI to correct undersizing and malapposition.

Optimal management of stent thrombosis has not been well defined in randomised trials due to the rarity of this complication and the fact that it consists a medical emergency.⁶ According to current guidelines routine

deferred stenting and routine thrombus aspiration should be avoided in the setting of STEMI.⁷ However, in the case of stent thrombosis high burden of thrombus, lack of information regarding the size and the type of the thrombosed stent and unawareness of the underlying mechanism in the acute setting, as in our case, could be preventing factors for deployment of an additional stent. Thrombus aspiration has shown beneficial clinical outcomes in stent thrombosis management⁸ and should be considered in these cases. In a large registry of patients with stent thrombosis around 50% of cases were treated with repeat stenting.⁹ Interestingly, in a more recent study that used OCT-guided PCI to evaluate and treat stent thrombosis, only 31% of the patients were treated with redo stent implantation⁴, suggesting that adjunctive intracoronary imaging may reduce unnecessary repeat stenting that might increase risk of restenosis and reocclusion.¹⁰ In addition, intensive antithrombotic pharmacotherapy has been used in the majority of reported cases of stent thrombosis^{4, 9} and in our case appeared effective in reducing thrombotic burden.

Identifying the underlying mechanism of stent thrombosis is of major importance as it can guide management strategy. In case of a malapposed stent, balloon angioplasty could be sufficient treatment, while in case of stent edge dissection an additional stent could be required. Intravascular imaging can both provide vital information regarding different mechanisms of stent thrombosis and successfully guide the subsequent corrective intervention. OCT provides higher resolution compared to intravascular ultrasound and is considered superior for detecting responsible mechanisms of thrombosis.¹¹ Use of OCT in a cohort of patients with stent thrombosis identified the underlying mechanism in 97% of the cases.⁴ In our case, OCT examination identified stent undersizing and malapposition as the most probable mechanism for the subacute stent thrombosis during the revision coronary angiogram. Balloon angioplasty was used to correct mechanical abnormalities and a final OCT control verified a satisfactory result. Finally, knowledge of previous stent sizes and implantation technique is necessary to understand limitations and determine realistic objectives for the corrective PCI.

Although management of stent thrombosis is challenging, a stepwise approach aiming firstly to stabilize the patient and restore flow TIMI III in the culprit artery and secondly to identify and treat the underlying mechanism of stent thrombosis should be considered.

Conclusion

In case of stent thrombosis, the objective should be to restore TIMI III flow and reverse the thrombogenic milieu,

while deferral of repeat stenting should be considered, especially when there are possible issues regarding previous stents. Intravascular imaging guidance during a revision procedure offers better understanding of issues with previous stents (such as undersizing, underexpansion, malapposition) that may contribute to stent thrombosis and accordingly plan a corrective PCI. Further data are warranted to help establish optimal treatment strategies in the occurrence of stent thrombosis.

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