Two Different Mechanisms of Very Late Stent Thrombosis: Assessment and Guidance of Therapy with Optical Coherence Tomography

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Two Different Mechanisms of Very Late Stent Thrombosis: Assessment and Guidance of Therapy with Optical Coherence Tomography

C. Roth, C. Gangl, G. Kreiner, G. Delle-Karth

Abstract: Incomplete coverage of stent struts and atherosclerosis within the neo-intima are the two most frequently mechanisms of very late stent thrombosis (VLST). As the determination by conventional coronary angiography is not always possible, the use of optical coherence tomography (OCT) is getting more and more important. The high resolution of OCT allows a closer look to the pathomechanisms of VLST, which helps to make the right decisions in the catheterization laboratory. The cases demonstrate the evaluation of VLST by OCT and the resulting therapeutic decisions.

Key words: very late stent thrombosis, mechanisms of stent thrombosis, drug eluting stents, optical coherence tomography


Schlüsselwörter: sehr späte Stentthrombose, Mechanismus der Stentthrombose, Medikamentenbeschichtete Stents, optische Kohärenztomographie

Introduction

Very late stent thrombosis (VLST) is a rare but potentially catastrophic event. With an annual occurrence rate of 0.6% [1] it has emerged as an important complication of percutaneous coronary intervention (PCI) with drug eluting stents (DES) [2, 3]. Two main risk factors for VLST were identified: First, incomplete coverage of stent struts and second atherosclerosis within the neo-intima. Whereas delayed healing with a higher percentage of uncovered stent struts is the key factor for thrombus formation in the former [4, 5], the rupture of a vulnerable plaque within the stent and potential vessel closure form the pathological substrate for the latter. These different mechanisms of VLST are frequently difficult to assess by coronary angiography alone. Optical coherence tomography (OCT) is a valuable tool in this setting [6, 7]. We herein report on two cases with VLST after DES for which we were able to assess the underlying pathomechanism and to guide therapeutic decisions by OCT.

Case 1

A 65-year-old man with a medical history of hyperlipidemia and hypertension underwent PCI in May 2005 because of acute anterior myocardial infarction. A 3.0 × 28 mm sirolimus eluting stent (Cypher Select®, Cordis Corporation, Johnson & Johnson, Warren, NJ) was implanted in the proximal LAD. The patient was prescribed aspirin (100 mg/day) for long term inhibition therapy (DAPT) was recommended. The peak troponin T concentration was measured on day 1 after infarction (0.43 ng/ml [normal range < 0.03]). The subsequent clinical course was uneventful and the patient left the hospital on day three after the stent thrombosis. Dual platelet inhibition therapy (DAPT) was recommended.

Case 2

A 48-year-old man underwent PCI in January 2007 with a paclitaxel eluting stent (Taxus liberté®, Boston Scientific Corpora-
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Discussion

These cases represent typical examples for the two main mechanisms for VLST, in which OCT was not only helpful in establishing the diagnosis but also supported the interven-
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With regard to delayed stent-endothelialization as underlying mechanism for VLST, recent OCT studies have underlined the importance of the percentage of uncovered stent struts and stent-vessel malapposition [8]. A significant gap between the stent and the arterial wall not only hampers adequate stent endothelialization but also promotes a prothrombotic milieu mainly because of the turbulent flow with subsequent low arterial wall shear stress [9, 10]. In our first patient 16% of all struts were uncovered even more than 5 years after the index procedure. Non-cover- age was predominantly detected at the malapposed proximal third of the stent. In line with the mild underexpansion of the stent at this site, the malapposition was probably related to the index procedure and not a result of positive remodeling. The critical question remains how to proceed with the patient during the acute PCI and in long term. With the help of the OCT findings we decided to only dissolve the thrombi and to place the patient on DAPT with aspirin and clopidogrel again. Alternative solutions would have been to perform high-pressure balloon angioplasty with a non-compliant balloon at the site of the malapposition. Nevertheless one must be aware that due to closed cell design of the Cypher® Stent a balloon-angioplasty might not be as effective as with newer generation DES and that a new vessel trauma because of balloon angioplasty could...
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potentially trigger further clinical events. Since healing of the stent struts cannot be expected in the future, any complete interruption of DAPT again puts the patient at an increased risk of stent thrombosis and whenever possible surgical interventions should at least be performed under the condition of continuous aspirin administration. If the surgical bleeding risk is estimated too high an alternative approach could be to stop the oral antiplatelet therapy but to install an infusion with a short acting GPIIb/IIIa inhibitor prior to surgery instead and to restart it in the postoperative phase.

In contrast to VLST caused by incomplete stent healing, in-stent neoatherosclerosis seen in DES [11, 12] is characterized by excessive neointima formation and accelerated atherosclerosis. These dynamic changes are probably induced by pro-inflammatory and/or hypersensitivity responses after the DES implantation. The OCT findings of our second patient showed important elements of unstable atherosclerotic plaque: a thin and ruptured fibrous cap, adherent thrombi and a dense intramural formation compatible with intramural hematoma and/or necrotic lipid rich core. In accordance with these OCT findings we decided to place a newer generation stent at the site of the plaque rupture.

**Conclusion**

Our cases demonstrate that with the help of high resolution OCT the differentiation of the two main mechanisms for VLST can be performed with high accuracy. This differentiation and detailed insights in the underlying pathomechanism may have an important impact on interventional and clinical decision-making.

**Conflict of Interest**

The authors declare that they have no conflict of interest.

**References:**

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