

## Clinical Image

# Very Late Stent Thrombosis Induced by Neoatherosclerosis 6 Years after Paclitaxel Eluting Stent Implantation: Optical Coherence Tomography Imaging

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## CLINICAL IMAGE

A 60-year-old man with history of hypertension, dyslipidemia, smoking, prior acute myocardial infarction and percutaneous coronary intervention (PCI) in 2010 to the mid left anterior descending (LAD) with 3.0×20mm paclitaxel eluting stent (PES; TAXUS, Boston Scientific Corporation, Natick, Massachusetts) was admitted with a ST - segment elevation myocardial infarction. Coronary angiography showed occlusion and thrombus at the site of stent previously implanted in LAD. After thrombus aspiration, optical coherence tomography (OCT) reveals an in -stent Neoatherosclerosis with intraluminal thrombus. The fibroatheroma had a lipid rich necrotic core. Plaque rupture was probably located behind wire artifact. Malapposed struts and uncovered struts were not detected (Figure 1). Following OCT, PCI was successfully performed with a scoring balloon (Scoreflex 3.5×10mm, OrbusNeich, Tokyo, Japan).

In - stent neoatherosclerosis is the development of atherosclerotic change in the neointimal tissue within previously implanted stent. It is an important mechanism for late stent failure such as in - stent restenosis and late stent thrombosis for both bare metal stent (BMS) and drug eluting stent (DES), especially in the late phase [1]. Pathologically, neoatherosclerosis is recognized as peristrut lipid - laden foamy macrophage clusters within neointima with or without calcification, necrotic core, fibroatheromas, thin - cap fibroatheromas (TCFA), and ruptures with thrombosis. TCFA is characterized by a necrotic core with an overlying fibrous cap measuring <65 μm, containing rare smooth muscle cells but numerous macrophages [2]. OCT allows a correct identification of all these findings: in particular, fibroatheromas defined as low signal zone with poorly delineated borders and a fibrous cap, whose thickness could be easily measured. Moreover, the OCT resolution (10 micron) allows identification of TCFA rupture with superimposed thrombus, characterized as a mass attached to luminal surface or floating within the lumen [3].

One histopathologic study showed that 1st generation DES develop neoatherosclerosis rapidly and more frequently

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Submitted: 11 August 2016

Accepted: 29 August 2016

Published: 30 August 2016

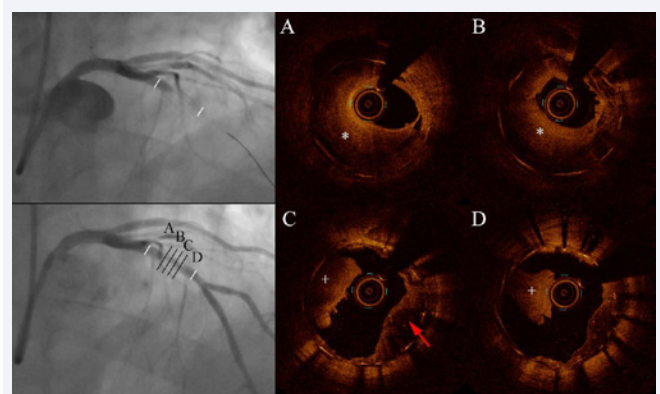
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compared with BMS, and no neoatherosclerosis was identified in BMS implanted ≤ 2 years. It is suggested that accelerated neoatherosclerosis in 1st generation DES might be caused by incompetent regenerated endothelium with poor cell-to-cell junctions that characterize impaired endothelial barrier function [2]. In addition, histologic study of 1st generation DES have demonstrated evidence of continuous neointimal growth during long-term follow-up, which is called as "late catch-up" phenomenon [1].

In the present report, we demonstrate the case of very late stent thrombosis induced by neoatherosclerosis 6 years after PES implantation. A recent OCT study suggested that time from stent implantation, drug eluting stent, active smoking, chronic kidney



**Figure 1** Left coronary artery angiography before (left upper-side) and after (left lower-side) thrombectomy (white bars indicate location of stent; black bars indicate OCT imaging sites). OCT findings: (A, B) Fibroatheroma was demonstrated as signal - poor and poorly delineated region (asterisk) (C, D) Intra luminal thrombus was demonstrated as irregular mass with attenuation (plus sign). Fibroatheroma was demonstrated as signal - poor region (red arrow). The cavity of the fibro atheroma is mostly empty, as necrotic core went inside the lumen through the ruptured cap. Ruptured cap is probably located behind the shadow of the wire.

Abbreviation: OCT: Optical Coherence Tomography

disease, and use of angiotensin converting enzyme inhibitors/angiotensin II receptor blockade were identified as independent predictors of neoatherosclerosis [4]. The patient was also current smoker. We should emphasize the risk of continuous smoking after stent implantation.

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### Cite this article

Ishida K, Ortega-Paz L, Brugaletta S, Sabaté M (2016) Very Late Stent Thrombosis Induced by Neoatherosclerosis 6 Years after Paclitaxel Eluting Stent Implantation: Optical Coherence Tomography Imaging. *JSM Atheroscler* 1(2): 1007.