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

Ishida K, Ortega-Paz L, Brugaletta S, and Sabaté M*
Department of Cardiology, University of Barcelona, Spain

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 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...

REFERENCES

