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# IMAGING VIGNETTE

## Clinical Utility of Combined Optical Coherence Tomography and Near-Infrared Spectroscopy for Assessing the Mechanism of Very Late Stent Thrombosis

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**VERY LATE STENT THROMBOSIS (VLST) IS A QUITE RARE BUT SERIOUS COMPLICATION THAT OFTEN** results myocardial infarction or cardiac death. In various cases of VLST (**Figures 1 to 4**), neoatherosclerosis with neointimal rupture has been shown to be a major contributor to VLST. Histologically, neo-atherosclerosis is characterized by accumulation of lipid-laden foamy macrophages within the neointima with or without necrotic core formation (1). Optical coherence tomography (OCT) is capable of detecting not only neoatherosclerosis characterized by lipid-laden neointima but also thrombus formation and/or neointimal rupture. However, there are concerns with OCT regarding the potential overdiagnosis of neoatherosclerosis (1). OCT can misjudge the neointima covered by thrombus as lipid-laden neointima. Near-infrared spectroscopy (NIRS) is the established imaging device that can discriminate lipid-laden plaque with high sensitivity and specificity by analyzing light absorption of coronary tissue components. In addition, a previous study (2) demonstrated the ability of NIRS-intravascular ultrasound to detect lipid-laden neointima within a stent. Findings on combined OCT and NIRS can provide the precise mechanism of VLST in vivo.

Manuscript received June 26, 2017; revised manuscript received October 31, 2017, accepted November 9, 2017.

From the Department of Cardiovascular Medicine, Wakayama Medical University, Wakayama, Japan. Dr. Kubo has received lecture fees from St. Jude Medical and Terumo. Dr. Shiono has received consulting fees from Philips Volcano. Dr. Akasaka has received lecture fees from St. Jude Medical, Terumo, and Abbott Vascular; and research grants from St. Jude Medical, Terumo, and Abbott Vascular. All other authors have reported that they have no relationships relevant to the contents of this paper to disclose.

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### FIGURE 1 Case 1: Neointimal Rupture With Neoatherosclerosis After BMS Implantation

A 73-year-old man had a history of undergoing a bare-metal stent (BMS) implantation in the proximal left anterior descending artery due to ST-segment elevation acute myocardial infarction. One hundred and thirty-two months after BMS implantation, he was admitted to our hospital due to anterior ST-segment elevation acute myocardial infarction. Emergency coronary angiography showed total thrombotic occlusion within BMS (A). Optical coherence tomography after thrombectomy showed neoatherosclerosis including thin-cap fibroatheroma-like neointima (a, \*), lipid-laden neointima (**b**, \*), neointimal rupture (**b** and **b**', red arrowhead), macrophage accumulation (a, blue arrowheads), and thrombus (b and c, white arrowheads) within BMS. Near-infrared spectroscopy chemogram (C) and near-infrared spectroscopy-intravascular ultrasound images (a' to c') showed neointima with high lipid burden (max lipid core burden index [LCBI]<sub>4mm</sub>; 829) within BMS. (A previous study has reported that the reference value of max LCBI<sub>4mm</sub> within freshly implanted stent for atherosclerotic lesion was 155  $\pm$  203 [2].) Final coronary angiography after balloon angioplasty showed no significant stenosis within BMS (B).



#### FIGURE 2 Case 2: Neointimal Rupture With Neoatherosclerosis After BMS Implantation

A 72-year-old man had a history of undergoing a BMS implantation in the mid left circumflex artery due to stable angina pectoris. Two hundred and eight months after BMS implantation, he was admitted to our hospital due to posterior ST-segment elevation acute myocardial infarction. Emergency coronary angiography showed subtotal thrombotic occlusion within BMS (A). Optical coherence tomography after thrombectomy showed neoatherosclerosis including lipid-laden neointima (a to c and a', \*), neointimal rupture (b, b', and c, red arrowheads), and thrombus (c, white arrowheads) within BMS. Near-infrared spectroscopy chemogram (C) and near-infrared spectroscopy-intravascular ultrasound images (a' to c') showed neointima with high lipid burden (max LCBI4mm; 810) within BMS. Final coronary angiography after balloon angioplasty and an everolimus-eluting stent (2.5  $\times$  24 mm) implantation showed no significant stenosis within BMS (B). Abbreviations as in Figure 1.

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