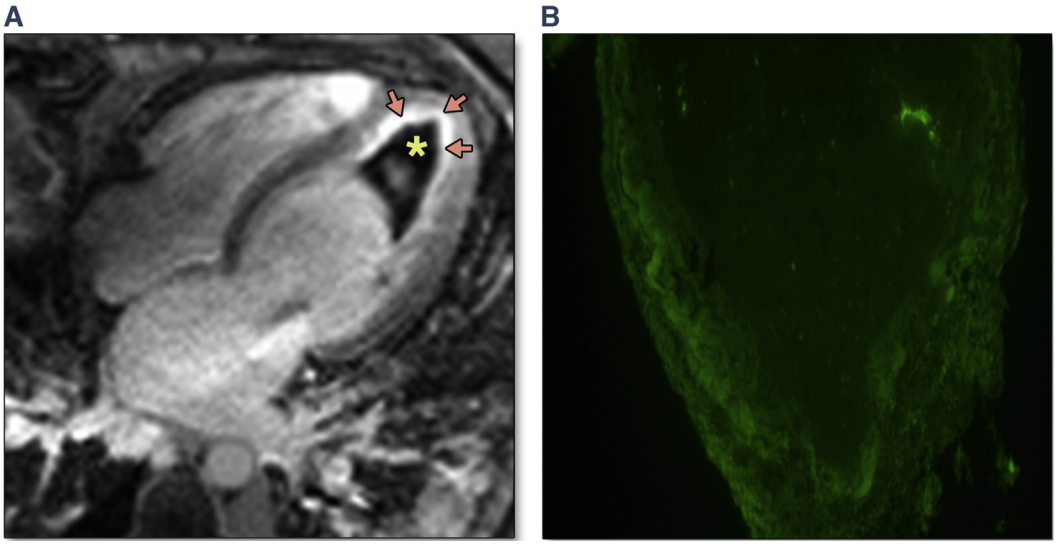
FIGURE 1 CMR and Immunohistochemical Stain



Phase-sensitive inversion recovery cardiac magnetic resonance (CMR) **(A)** demonstrates an apical thrombus **(*)** and delayed enhancement of the endomyocardial border **(arrows)**, suggesting endomyocardial fibrosis or inflammation. Endomyocardium with immunohistochemical staining against eosinophil-derived neurotoxin **(B)**.

may not even have eosinophilic infiltration on EMB (2). In patients without peripheral eosinophilia or eosinophilic tissue infiltrate on EMB, positive immunohistochemical staining with polyclonal antibodies against eosinophil major basic protein 1 and eosinophil-derived neurotoxin can support the diagnosis. Dimethyl fumarate (Tecfidera) should probably be considered as an etiologic agent for eosinophilic cardiac injury in appropriate situations.

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Strut Coverage After Paclitaxel-Eluting Stent Implantation in the Superficial Femoral Artery

In the superficial femoral artery (SFA), quantitative analysis for vascular healing response after self-expanding nitinol bare metal stent (BMS) or drug-eluting stent (DES) implantation is limited. The purpose of this study was to evaluate the endothelial strut coverage after nitinol paclitaxel-eluting stent (PES) and BMS implantation in the SFA with optical frequency domain imaging (OFDI).

This was a prospective, randomized, open-label study to investigate vascular healing response to PES (Zilver PTX Cook Medical, Bloomington, Indiana) and BMS (S.M.A.R.T. Control, Cordis, Miami Lakes, Florida). A total of 40 SFA lesions suitable for the OFDI procedure were randomized 1:1 to receive either PES or BMS implantation and the vascular healing response using OFDI 6 months after stenting was evaluated. Aspirin 100 mg/day and clopidogrel 75 mg/day were continued during the follow-up period.

The OFDI procedure was performed with a Terumo OFDI system (FastView, Terumo Corporation, Tokyo, Japan) using a nonocclusive technique at an image

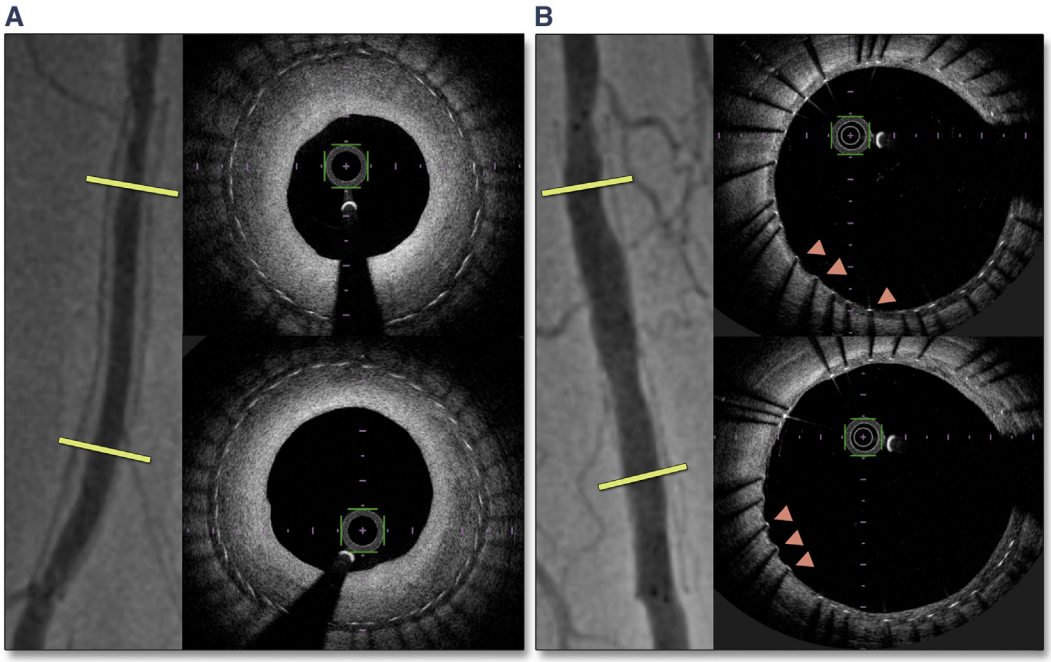
acquisition rate of 160 frames/s. After insertion of a 6-F guiding sheath through an ipsilateral or contralateral femoral artery, the guiding sheath was advanced past the common femoral artery and positioned 10 to 20 mm proximal to the stent edge. Automated OFDI pullback at a speed of 40 mm/s was performed during continuous injection of 50% contrast medium with a flow rate of 8 ml/s from the guiding sheath with simultaneous manual obstruction of the common femoral artery over the guiding sheath.

Cross-sectional OFDI images were analyzed at 5-mm intervals (every 20 frames). A total of 611 cross-sectional images were reviewed. Among those, 23 (3.8%) were excluded from the analysis because of insufficient image quality mainly caused by residual blood. Stent strut was defined as uncovered if any part of the strut was visibly exposed to the lumen. The number of uncovered struts and total struts per lesions were counted, and the percentage of uncovered struts was calculated as a ratio of uncovered struts to total struts. If neointimal coverage on the strut was observed, neointimal tissue (NIT) thickness was measured. The primary endpoint of this study was to compare the percentage of uncovered stent struts between BMS and PES at 6-month follow-up.

Among the 40 lesions, 2 patients in the BMS group died and 1 patient in the PES group required target lesion revascularization due to recurrent symptoms during the follow-up period. No stent thrombosis was observed. Finally, follow-up OFDI was performed for 16 lesions in the BMS group and 16 lesions in the PES group, and 14,959 stent struts were analyzed. The total stent length was 96.5 ± 29.1 mm in the BMS group and 81.5 ± 37.6 mm in the PES group ($p = 0.17$). Intraclass correlation coefficients for intraobserver and interobserver reliability of NIT thickness derived from a total of 100 randomly selected cross sections were 0.998 and 0.997, respectively. In a strut-level analysis, mean NIT thickness was less in the PES group compared with the BMS group (436 ± 134 μm vs. 665 ± 163 μm, $p < 0.001$). The percentage of uncovered struts per lesion was significantly higher in the PES group (13.6% vs. 2.4%, $p < 0.01$) (Figure 1).

Similar to the reports on the coronary artery, the percentage of uncovered struts was higher after DES implantation compared with BMS in the SFA. Although the use of DES has reduced the rates of restenosis and late lumen loss in the coronary artery, life-threatening complications of this technology, such as late stent thrombosis (LST), have emerged as

FIGURE 1 OFDI Findings in Representative Cases of Vascular Response After Nitinol BMS and Nitinol PES Implantation



(A) OFDI of stent struts surrounded by a thick neointimal formation at 6-month BMS follow-up. **(B)** OFDI of uncovered struts **(arrowheads)** at 6-month PES follow-up. BMS = bare metal stent(s); OFDI = optical frequency domain imaging; PES = paclitaxel-eluting stent(s).

a major concern. Lack of healing and absence of endothelial cell coverage of the stent struts of DES have been strongly associated with LST in human autopsy studies (1). Similar to the coronary artery, stent thrombosis due to uncovered struts should also be a concern after DES implantation in the peripheral artery. A recently published case report described LST in the SFA after Zilver PTX implantation (2). According to the case report, LST occurred due to discontinuation of an antiplatelet agent. Uncovered stent struts with a large red thrombus were documented in the Zilver PTX by angioscopy. To date, there are no standard guidelines or consensus for antiplatelet therapy after DES implantation in the femoropopliteal artery lesion. Therefore, prospective, randomized studies are required to confirm the optimal antiplatelet therapy after DES implantation.

In conclusion, OFDI revealed that vascular healing after DES implantation in the SFA was impaired during the long-term phase. (Proteomic analysis of cases with pancreatic cancer who undergo preoperative chemoradiation therapy; UMIN000014698).

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Whole-Body Visualization of Ectopic Bone Formation of Arteries and Skin in Pseudoxanthoma Elasticum

Pseudoxanthoma elasticum (PXE), CD73 deficiency, generalized arterial calcification of infancy, and progeria involve accelerated medial arterial calcification (MAC) leading to premature cardiovascular morbidity and mortality. MAC is also observed in diabetes mellitus, chronic kidney disease, and the elderly (1), and insufficient levels of inorganic pyrophosphate are thought to be involved. Finding a therapy for the genetic syndromes may cure these diseases and provide a model for lowering cardiovascular disease burden in the aging population. Drug discovery would be facilitated by a powerful intermediate endpoint. Positron emission tomography/computed tomography (PET/CT) with radioactive sodium fluoride (^{18}NaF) provides quantitative information on arterial calcification formation. We aimed to provide proof-of-concept that whole-body deregulated calcification metabolism in PXE could be visualized and quantified with ^{18}NaF PET/CT.

In our clinic we investigated 4 female patients with PXE using ^{18}NaF PET/CT. Image acquisition was done on a whole-body LSO PET scanner with a 40-slice CT scanner 90 min after injection of 2.0 MBq/kg ^{18}NaF. Low-dose CT involved 100 to 120 kV, 8 to 155 mA (combined effective dose, 5 mSv). On ^{18}NaF PET/CT, we measured femoral artery and skin findings. Briefly, for arteries, an observer drew regions of interest around the arteries and in the right atrium to derive a corrected maximal standardized uptake value ($cSUV_{max}$) and a target-to-blood pool ratio (TBR). On CT, Agatston-like calcium scores of lesions with >130 Hounsfield units (HU) in the arteries and the bone density in the lumbar spine were quantified. Skin abnormalities in the neck, axillar, and inguinal regions were visually scored as absent, mild, moderate, or severe by 1 observer at standard window settings (CT, 1500/450; PET, count per megabecquerel of 459/2,481 [IntelliSpace Portal client version 4.0, Philips Medical Systems, Eindhoven, the Netherlands]). The same regions were evaluated by a physician blinded to the PET/CT images by using the Phenodex classification (2).

The patients were 43, 49, 57, and 67 years of age, respectively. On CT, we visually identified long segments with thin circular calcifications, predominantly in the femoral arteries (**Figure 1**). Interestingly, the density of these calcifications was <130 HU, and, therefore, the software could not calculate Agatston scores. Visually, especially in the femoral arteries, the