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).

Kojiro Miki, MD
Kenichi Fujii, MD*
Masashi Fukunaga, MD
Machiko Nishimura, MD
Tetsuo Horimitsu, MD
Ten Saita, MD
Akironi Sumiyoshi, MD
Hirotu Tamaru, MD
Takihiro Imanaka, MD
Masahiko Shibuya, MD
Yoshio Naito, MD
Tohru Masuyama, MD
Masaharu Ishihara, MD
*Cardiovascular Division
Hyogo College of Medicine
1-1 Mukogawa-cho
Nishinomiya-city, Hyogo 663-8501
Japan
E-mail: kfuji@hyo-med.ac.jp

Please note: The authors thank the staffs in the catheterization laboratory at Hyogo College of Medicine for their excellent assistance during the study. The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

REFERENCES

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 (18NaF) 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 18NaF PET/CT.

In our clinic we investigated 4 female patients with PXE using 18NaF 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 18NaF. Low-dose CT involved 100 to 120 kV, 8 to 155 mA (combined effective dose, 5 mSv). On 18NaF 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 (cSUVmax) 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
arterial $^{18}$NaF signal was increased at levels similar to those for cortical bone. In the right femoral artery, the cSUV$_{\text{max}}$ was 0.2, 0.2, 2.1, and 1.1, and the TBR was 1.4, 1.3, 3.3, and 1.9; in the left femoral artery, the cSUV$_{\text{max}}$ was 0.2, 0.1, 2.2, and 1.3; and the TBR was 1.5, 1.1, 3.4, and 2.1 for the 4 patients with increasing age, respectively. On CT, the HU value in the lumbar vertebra 1 was 252, 169, 82, and 122, respectively. Values <120 are considered osteoporosis. Regarding the skin, patient 1 had severe skin abnormalities, both clinically and on PET, patient 2 had mild skin abnormalities both clinically and on PET, patient 3 had moderate skin abnormalities clinically and on PET, and patient 4 had moderate skin abnormalities clinically and severe on PET. Skin abnormalities were identified at all locations, and the severity and location of PET scores corresponded favorably with the clinical classification ($R_{\text{spearman}} = 0.44, p = 0.09$).

In conclusion, this is the first report showing that $^{18}$NaF PET/CT is able to visualize and quantify ectopic calcifications and bone metabolism in PXE and may prove a valuable intermediate mechanistic endpoint for therapeutic studies aiming to slow or remove MAC.

Sytse F. Oudkerk, MD
Pim A. de Jong, MD, PhD*
Björn A. Blomberg, MD
Asbjørn M. Scholtens, MD
Willem P.Th.M. Mali, MD, PhD
Wilko Spiering, MD, PhD
*Department of Radiology and Nuclear Medicine
University Medical Center Utrecht
Room E01.132
Heidelberglaan 100
3508 GA Utrecht
the Netherlands
E-mail: p.dejong-8@umcutrecht.nl
http://dx.doi.org/10.1016/j.jcmg.2015.06.006

Please note: The authors have reported that they have no relationships relevant to the contents of this paper to disclose.

REFERENCES

Heart Rate Is an Important Consideration for Cardiac Imaging of Diastolic Function

We read with considerable interest the state-of-the-art paper, “Cardiac Imaging to Evaluate Left Ventricular Diastolic Function,” by Flachskampf et al. (1). The authors provide comprehensive coverage of the methods used to image and analyze diastolic function.

For 2 reasons, heart rate merits further discussion. 1) There is significant translational interest in diastolic function. It is sometimes underappreciated that, in the mouse and rat models, one cannot separate early diastolic and late atrial filling without significantly reducing the heart rate. 2) In humans, it is not possible to separate early diastolic and late atrial filling modestly at fast heart rates ($\geq$100 beats/min).

Figure 1A shows the importance of heart rate. Systolic duration was reduced only modestly, and E-wave peak velocity and timing were essentially unchanged as the heart rate increased. Atrial filling merges (fusion) with the E-wave until it was impossible to differentiate. The atrial contribution to filling is nearly preserved, simply adding to the E-wave (2).