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POSITRON EMISSION TOMOGRAPHY TO IDENTIFY RUPTURED AND VULNERABLE CORONARY PLAQUES

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Background Non-invasive imaging to identify vulnerable or ruptured coronary artery plaque would represent a major clinical advance. Using positron emission tomography (PET) and computed tomography (CT), we investigated coronary uptake of 18F-fluoride (18F-NaF) and 18F-fluorodeoxyglucose (18F-FDG) in patients with acute myocardial infarction or stable angina.

Methods Forty patients with acute myocardial infarction and 40 with stable angina underwent electrocardiogram-gated 18F-NaF and 18F-FDG PET-CT and invasive coronary angiography. 18F-NaF uptake was compared with virtual histology intravascular ultrasound in patients with stable angina, and with histology in 12 carotid endartectomy specimens.

Results Intense focal 18F-NaF uptake occurred at the site of plaque rupture in 37 (93%) patients with myocardial infarction (tissue-to-background ratio [TBR], 1.66 [1.40-2.25] versus 1.24 [1.06–1.38]; culprit versus maximal non-culprit, P<0.001). In patients with stable angina, 18 (45%) had focal plaque 18F-NaF uptake (2.10 [1.71-2.81]) that, compared to plaques without uptake, had more high-risk features: positive remodeling (vessel area 24 [17-27] versus 14 [12-18] mm2; P=0.002), necrotic core (24.6% [20.5-28.8] verses 18.0% [14.0-22.4], P=0.001) and microcalcification (73 versus 21%, P=0.002). Carotid plaque rupture also co-localized with ex vivo 18F-NaF uptake and was associated with areas of apoptosis, necrosis and active calcification. Myocardial uptake markedly hampered 18F-FDG assessment in most patients (55%) and even where coronary uptake was discernible, there were no differences between culprit and non-culprit lesions (1.71 [1.40-2.13] versus 1.58 [1.28-2.01]; P=0.34).

Conclusions 18F-NaF holds major promise as a novel biomarker of coronary plaque vulnerability and rupture with implications for the diagnosis, investigation and treatment of coronary artery disease.