C

POSITRON EMISSION TOMOGRAPHY TO IDENTIFY RUPTURED AND VULNERABLE CORONARY PLAQUES

N V Joshi,[1] A Vesey,[1] F H M Craighead,[1] M C Williams,[1] S E Yeoh,[1] A S Shah,[1] A Fletcher,[1] A D Flapan,[1] P Calvert,[2] E J R van Beek,[1] M Behan,[1] N Cruden,[1] N G Uren,[1] D Berman,[1] N L Mills,[1] J H F Rudd,[2] M R Dweck,[1] D E Newby[1] *[1]Centre for Cardiovascular Science / Clinical Research Imaging Centre University of Edinburgh / Edinburgh Heart Centre; [2]Division of Cardiovascular Medicine, University of Cambridge, UK (PC, JHFR) and Cedars Sinai Medical Centre, Los Angeles (DB)*

doi:10.1136/heartjnl-2013-304019.276

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.