

Fusion of positron emission tomography and coronary computed tomographic angiography identifies fluorine 18 fluorodeoxyglucose uptake in the left main coronary artery soft plaque

Erick Alexanderson, MD,^{a,b} Piotr Slomka, PhD,^{c,d,e} Victor Cheng, MD,^{c,d,e}

Aloha Meave, MD,^{a,b} Yolanda Saldaña, MD,^a Leonardo García-Rojas, MD,^a and Daniel Berman, MD^{c,d,e}

Case report. A 71 year-old man underwent F-18 FDG-PET scanning with noncontrast computed tomography (CT) for attenuation and anatomic correction (PET/CT) 1 year after surgical resection with diverting colostomy and adjuvant chemotherapy and radiotherapy for colonic adenocarcinoma. A PET/CT scan was done after 10 hours of fasting, 90 minutes after FDG injection. His medical history included type 2 diabetes mellitus controlled with metformin and glibenclamide, cigarette smoking for 15 years (8-10 cigarettes per day), chronic gastritis, heavy alcohol use, and esophageal varices. The patient denied any allergies, chest pain, or previous heart disease.

FDG-PET/CT showed focal hypermetabolic activity in the rectum and its neighboring fatty tissue, liver, and multiple lymph nodes in the mediastinum and neck. In addition, noticeable FDG uptake was present in the aorta, most consistent with aortic atherosclerotic plaque, and in a structure that appeared to be within or adjacent to the left main coronary artery (Figure 1).

To further investigate the latter finding, retrospectively gated 64-slice CCTA was performed on the same day with the Siemens HI-REZ Biograph 64 hybrid PET/CT scanner (Siemens Medical Solutions, Malvern, Pa). Analysis of the reconstructed 3-dimensional CCTA images identified significant coronary atherosclerosis, including a large noncalcified plaque in the left main

coronary artery and the proximal left anterior descending artery, the latter associated with a 50% stenosis, as well as multiple areas of calcified plaque in the right coronary artery (Figure 2). Subsequent software fusion of CCTA and FDG-PET/CT images by use of the Cedars-Sinai CT-Fusion option in the QPS program (Cedars-Sinai Medical Center, Los Angeles, Calif) showed anatomic correspondence between the abnormal extra-aortic FDG uptake and the noncalcified plaque within the left main coronary artery (Figure 3). In view of these findings, the patient was referred to the cardiology clinic, and we recommended aspirin and statin therapy and further examination by coronary angiography. However, because of his current serious oncologic condition, coronary angiography was not performed.

Discussion. There is mounting evidence that FDG-PET uptake reflects inflammation in atherosclerotic plaques. Animal studies with atherosclerotic rabbits have confirmed that FDG uptake corresponds with plaque macrophage content.^{1,2} In patients with angiographic evidence of internal carotid stenosis, Rudd et al³ found significantly higher FDG uptake in 8 symptomatic carotid plaques than in 6 contralateral asymptomatic plaques; normal carotid arteries exhibited no identifiable uptake. Major limitations of plaque imaging with FDG-PET in the coronary vessels include cardiac motion during PET, FDG uptake in adjacent structures such as the myocardium, and limited PET resolution. Despite these limitations, our case shows the ability of FDG-PET/CT and CCTA image fusion to identify and localize areas of noticeable FDG uptake in the proximal segments of the coronary arteries, which could be related to inflamed atherosclerotic lesions.

Acknowledgment

The authors thank Dr Paul Guillermo Mendoza Vasquez and Dr Pedro Alberto Lamothe Molina for their technical support. The authors have indicated they have no financial conflicts of interest.

From the Unidad PET/CT Ciclotron, Facultad de Medicina, Universidad Nacional Autónoma de México,^a and Instituto Nacional de Cardiología Ignacio Chávez, Mexico City, Mexico,^b and Departments of Medicine,^c and Imaging,^d Cedars-Sinai Medical Center, and Department of Medicine, David Geffen School of Medicine at the UCLA,^e Los Angeles, Calif.

Reprint requests: Erick Alexanderson, MD, Juan Badiano, No. 1 Colonia Sección XVI, CP 14080, Mexico City, Mexico; alexanderick@yahoo.com.

J Nucl Cardiol 2008;15:841-3.
1071-3581/\$34.00

Copyright © 2008 by the American Society of Nuclear Cardiology. All rights reserved.

doi:10.1016/j.nuclcard.2008.06.014

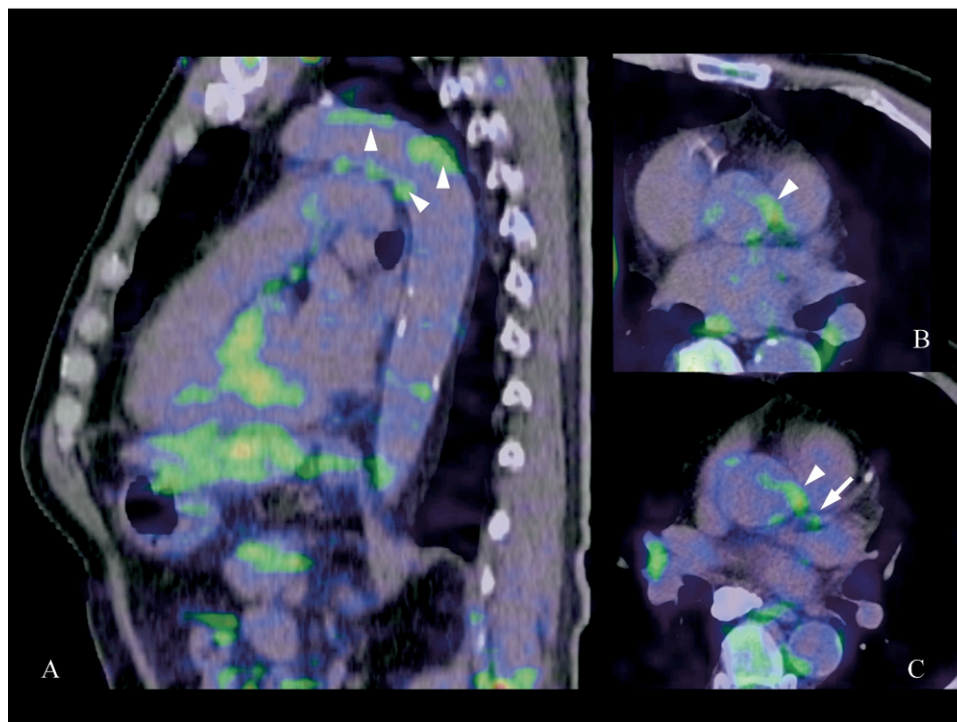


Figure 1. F-18 FDG-PET/CT images in sagittal (A) and axial (B and C) views showing uptake in aortic arch (*arrowheads* in A) and ascending aortic root (*arrowheads* in B and C), as well as a structure that appears to be within left main coronary artery (*arrow* in C). The maximal standard uptake values in the lesions were 2.5 in the aorta and 2.1 in the left main artery. In comparison, the maximal standard uptake value was 1.2 in the adjacent myocardial region.

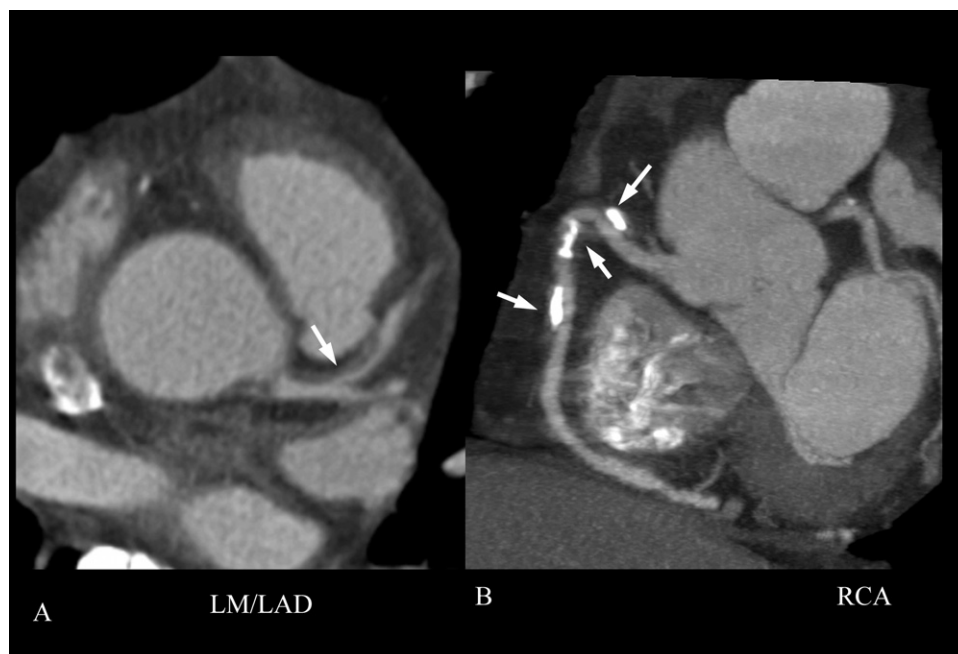


Figure 2. Representative CCTA images of left main coronary artery (LM) (A) and right coronary artery (RCA) (B), showing a large, radiopaque, noncalcified plaque in the body of the left main coronary artery (*arrow* in A) and multiple calcified plaques in the right coronary artery (*arrows* in B). LAD, Left anterior descending artery.

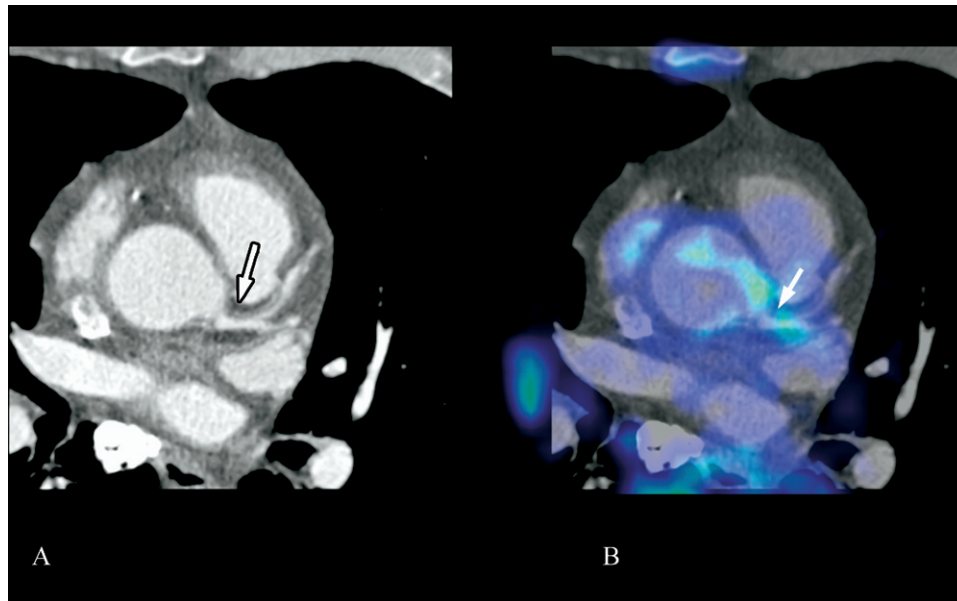


Figure 3. CCTA image of noncalcified plaque in left main coronary artery (A) (arrow) and corresponding image after fusion with F-18 FDG-PET/CT (B), localizing the inflammatory PET signal with a maximal standard uptake value of 2.1 to the noncalcified plaque seen in the left coronary artery (arrow).

References

1. Tawakol A, Migrino RQ, Hoffmann U, Abbara S, Houser S, Gewirtz H, et al. Noninvasive in vivo measurement of vascular inflammation with F-18 fluorodeoxyglucose positron emission tomography. *J Nucl Cardiol* 2005;12:294-301.
2. Ogawa M, Ishino S, Mukai T, Asano D, Teramoto N, Watabe H, et al. (18)F-FDG accumulation in atherosclerotic plaques: Immunohistochemical and PET imaging study. *J Nucl Med* 2004;45:1245-50.
3. Rudd JH, Warburton EA, Fryer TD, Jones HA, Clark JC, Antoun N, et al. Imaging atherosclerotic plaque inflammation with [18F]-fluorodeoxyglucose positron emission tomography. *Circulation* 2002;105:2708-11.