



Combined CT-PET criteria for myocardial viability and scar: a preliminary report

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Abstract

Identification of hibernating myocardium and its differentiation from scar tissue is an important clinical task for implementing different treatment options. The recognition of combined criteria in spiral-CT and PET for hibernating myocardium and scar will be important as hybrid CT-PET moves into the main stream of routine oncologic and cardiovascular imaging. A review of three cases, initially referred for CT evaluation of cardiac or pulmonary conditions with subsequent F-18 FDG body PET imaging, was presented for illustration as a combined CT-PET cardiac evaluation. The real value of this combined anatomical and functional evaluation will be with the upcoming concurrent CT and PET imaging using dedicated multi-slice CT-PET scanners.

Introduction

PET imaging using the concepts of perfusion and metabolism mismatch has been widely used in differentiating viable hibernating myocardium from myocardial scar [1–4]. An integrated approach of CT angiography with MRI viability assessment has been proposed [5]. As spiral CT becomes more available for evaluating cancer and coronary artery disease with F-18 FDG PET, it is important to recognize combined CT-PET criteria for hibernating myocardium and scar. To the best of our knowledge, there are no published data on combined CT-PET imaging for myocardial viability. We hypothesize that myocardial hypoenhancement by CT with concomitant FDG uptake by PET likely represents viable hibernating myocardium. We presented three cases to illustrate this hypothesis. The patients were initially referred for CT evaluation of cardiac or pulmonary conditions. They were later studied by F-18 FDG PET

imaging for oncologic evaluation of CT findings. It was at that time that the combined cardiac evaluation by CT-PET was additionally performed. The case reports emphasize primarily the cardiac history and myocardial findings by combined CT and PET imaging. Thus, many of the oncologic details are not reported.

Case reports

F-18 FDG PET images were acquired (in 256×256 matrix) with a dedicated whole body PET camera (GE ADVANCE, GE Medical Systems, Milwaukee, WI) with 370 MBq F-18 FDG 1 h after injection to the patient who fasted for 4 h at resting condition. The images were reconstructed by iterative reconstruction (128×128 matrix, 28 subsets, 2 iterations) with segmented attenuation correction. The spiral CT images were obtained shortly before the PET scan with a

multi-detector (4-slice) CT scanner (SIEMENS VOLUME ZOOM, Siemens Medical Systems AG, Germany) and 1 mm coronal and sagittal multi-planar reformations. CT scanning commenced 25–30 s following a peripheral i.v. injection (4 cc/s) of non-ionic contrast (175 cc OMNIPAQUE 350, Amersham Health, Princeton, NJ) via a power injector (Liebel-Florsheim, Cincinnati, OH) utilizing the following parameters: 3 mm beam collimation, 1.5 mm reconstruction, 120 kVp, 330–400 mAs, 512 × 512 matrix, 0.5 s rotation time. All cardiac segments in CT and PET were named according to standardized AHA segmentation nomenclature [6].

Case 1: Myocardial scar (Figure 1)

Fifty-five-year old man with history of coronary artery triple vessel disease, myocardial infarction and CABG was referred initially for evaluation of left ventricular mass. The cardiac catheterization showed 40% mid-left anterior descending, 60% first diagonal, 70% circumflex, 80% first obtuse marginal and 90% mid-right coronary arterial stenosis. Echocardiography showed lateral wall hypokinesis and left ventricular thrombus.

The CT scan showed a non-enhancing left ventricular mass consistent with left intraventricular thrombus and lateral subendocardial hypoenhancement and thinning (Figure 1A). F-18 FDG PET showed no metabolic uptake corresponding to the area of subendocardial hypoenhancement

and thinning seen on CT in the lateral wall indicating a region of non-viable myocardium or scar (Figure 1B).

Case 2: Hibernating myocardium (Figure 2)

Fifty-six-year old female was referred for pre-operative CT evaluation for her history of abdominal aortic aneurysm before vascular intervention. The CT study showed subendocardial hypoenhancement in the basal inferior wall with no myocardial wall thinning (Figure 2A and C). The F-18 FDG PET scan showed significant FDG uptake in the same basal inferior myocardial segment (Figure 2B and D), indicating viable hibernating myocardium as proven by the classic flow-metabolism mismatch pattern from the hypoperfusion in Tc-99m Sestamibi SPECT scan (Figure 2E) with corresponding enhanced metabolism in F-18 FDG PET scan (Figure 2D). The perfusion study was redundant but it was included for confirmation in the illustration.

Case 3: Mixture of scar and hibernating myocardium (Figure 3)

Seventy-six-year old man with history of severe triple vessel disease had myocardial infarction in the mid to basal inferolateral region before CABG. Cardiac catheterization showed an occluded saphenous graft to the circumflex artery. The left internal mammary artery (LIMA) graft to

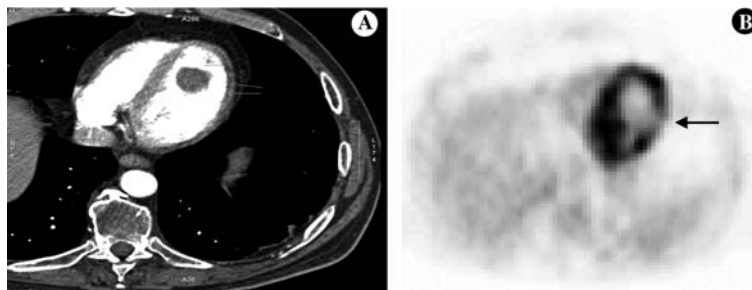


Figure 1. (A) Contrast enhanced axial CT image shows myocardial thinning and subendocardial hypoenhancement along inferolateral region (small arrows) with intraventricular thrombus. (B) Axial F-18 FDG PET images show a corresponding metabolic defect, viz. without significant FDG uptake along inferolateral area (single black arrow).

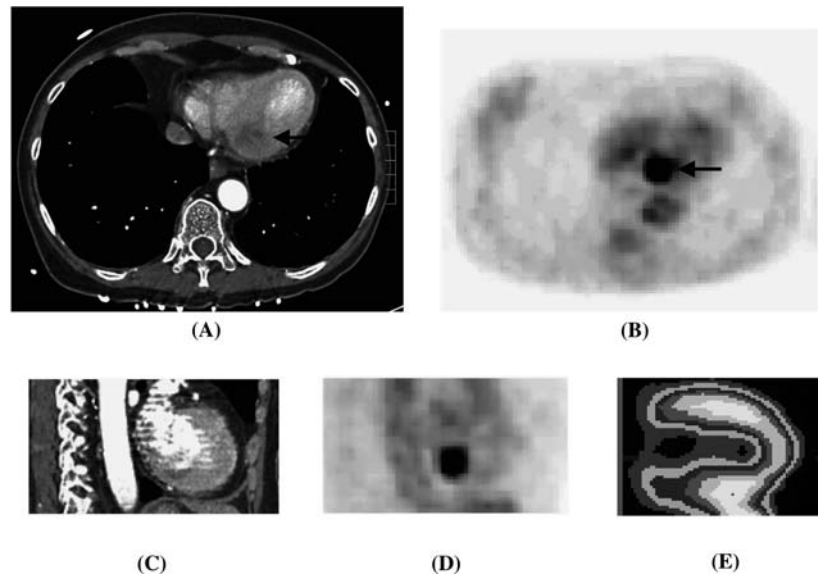


Figure 2. Contrast enhanced axial (A) and sagittal (C) multiplanar CT images show basal inferior subendocardial hypoenhancement without thinning (arrow). The respective F-18 FDG PET images (B and D) show intense uptake in the corresponding basal inferior region (black arrow), suggesting viable hibernating myocardium as confirmed by flow-metabolic mismatch on Tc-99m Sestamibi resting SPECT with severe basal inferior hypoperfusion (E).

the left anterior descending artery and saphenous graft to right coronary artery were both patent.

CT showed transmural hypoenhancement and myocardial thinning in the mid to basal inferolateral

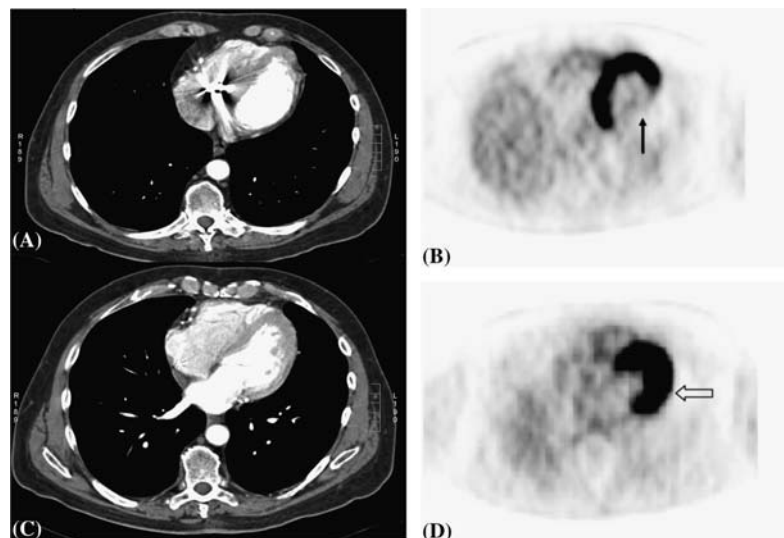


Figure 3. Contrast enhanced axial CT scan shows hypoenhancement with thinning of the mid to basal inferolateral myocardium (A). F-18 FDG PET scan shows a corresponding metabolic defect indicating presence of old infarction or scar (B, solid arrow). However, in the basal anterolateral region, there is hypoenhancement without significant thinning (C). The basal anterolateral region demonstrates FDG uptake indicating presence of viable hibernating myocardium (D, open arrow).

region (Figure 3A). Along the basal anterolateral region, there was subendocardial non-transmural hypoenhancement without significant myocardial thinning (Figure 3C).

The F-18 FDG PET showed a severe metabolic defect in the mid to basal inferolateral region, which coincided to the area of transmural hypoenhancement and myocardial thinning on CT, suggesting myocardial scar due to prior infarction (Figure 3B). But, along the basal anterolateral region where CT showed subendocardial hypoenhancement without thinning, there was significant FDG uptake by PET, indicating most likely viable hibernating myocardium (Figure 3D).

Discussion and conclusion

Identification of myocardial perfusion abnormalities as a result of myocardial infarction have been described with contrast enhanced CT [7–10]. The findings of hypoenhancement in regions of myocardial infarction are on the basis of markedly reduced myocardial blood flow (10–20% of normal myocardial perfusion) [11]. This 5- to 10-fold decrease in myocardial perfusion allows one to visualize contrast enhancement differences between normally perfused myocardium and infarcted non-viable myocardium on rest contrast enhanced imaging studies such as CT or MRI. In fact, in order to identify a perfusion abnormality, it is well known that a 2-fold vasodilated flow reduction is required [11]. As such, if there is a greater than 2-fold difference in myocardial perfusion, hypoenhancement should be identified on first pass contrast enhanced CT or MRI studies. Unfortunately, CT cannot quantify the degree of hypoenhancement and differentiate the infarcted non-viable myocardium from severely ischemic viable hibernating myocardium. This differentiation, however, can be established with combined CT and metabolic F-18 FDG PET imaging since viable hibernating myocardium is FDG avid whereas, non-viable infarcted tissue or scar is not [3].

The cases illustrated that the information on cardiac viability may be readily obtained by rou-

tine spiral CT and F-18 FDG PET. These may be useful preliminary findings for the future and have value in cardiac evaluation as concurrent anatomical (CT) and functional (PET) imaging by combined multi-slice spiral CT-PET scanners become widely available. We hypothesize the use of CT-PET scanners as a combined examination to provide information on myocardial hypoenhancement, thinning and FDG uptake for viability. We propose the following criteria: 'Myocardium that shows hypoenhancement and myocardial thinning by CT without FDG uptake by PET is likely due to scar, whereas if there is only hypoenhancement without myocardial thinning by CT but there is FDG uptake, it most likely represents viable hibernating myocardium.' With a combined CT-PET scanner, the patients referred for myocardial viability may be evaluated in a single setting for coronary anatomy by CTA, cardiac function and ejection fraction by gated wall motion, myocardial perfusion by multi-slice thin-cut contrast CT and myocyte function (metabolism) by FDG PET. If myocardial hypoenhancement cannot be detected by CT, then additional resting perfusion by PET is mandatory for evidence of perfusion abnormalities. The observations of the current reports re-iterate the important concept that enhanced FDG uptake by PET concomitant with any indications of severe reduction of perfusion either manifested by CT subendocardial hypoenhancement or PET myocardial hypoperfusion defects suggests the presence of viable hibernating myocardium. A large study is necessary to compare these criteria with traditional PET assessment of myocardial viability by perfusion and metabolism mismatch and with the ultimate gold standard of viability through functional recovery after revascularization. As cardiac diseases and cancers are the top two causes of death in developed countries, recognition of such CT-PET findings are important incidental findings during oncologic evaluation. These findings provide additional value during the 'one-stop' cardiac evaluation by combining non-invasive angiography from CT and gated wall motion with established methods of measuring metabolism and/or perfusion from PET for the assessment of viability.

References

1. Mari C, Strauss WH. Detection and characterization of hibernating myocardium. *Nucl Med Commun* 2002; 23(4): 311–322.
2. Maddahi J, Schelbert H, Brunken R, Di Carli M. Role of thallium-201 and PET imaging in evaluation of myocardial viability and management of patients with coronary artery disease and left ventricular dysfunction. *J Nucl Med* 1994; 35(4): 707–715.
3. Go RT, MacIntyre WJ, Cook SA, et al. The incidence of scintigraphically viable and nonviable tissue by rubidium-82 and fluorine-18-fluorodeoxyglucose positron emission tomographic imaging in patients with prior infarction and left ventricular dysfunction. *J Nucl Cardiol* 1996; 3(2): 96–104.
4. Bonow RO, Dilsizian V, Cuocolo A, Bacharach SL. Identification of viable myocardium in patients with chronic coronary artery disease and left ventricular dysfunction. Comparison of thallium scintigraphy with reinjection and PET imaging with 18F-fluorodeoxyglucose. *Circulation* 1991; 83(1): 26–37.
5. White RD, Setser RM. Integrated approach to evaluating coronary artery disease and ischemic heart disease. *Am J Cardiol* 2002; 90(10C): 49L–55L.
6. Cerqueira MD, Weissman NJ, Dilsizian V, et al. Standardized myocardial segmentation and nomenclature for tomographic imaging of the heart: a statement for health-care professionals from the Cardiac Imaging Committee of the Council on Clinical Cardiology of the American Heart Association. *Circulation* 2002; 105(4): 539–542.
7. Lipton MJ, Bogaert J, Boxt LM, Reba RC. Imaging of ischemic heart disease. *Eur Radiol* 2002; 12(5): 1061–1080.
8. Lipton MJ, Higgins CB. Evaluation of ischemic heart disease by computerized transmission tomography. *Radiol Clin North Am* 1980; 18(3): 557–576.
9. Godwin JD, Moore AV, Ideker RE, Califf RM. Prospective demonstration of myocardial infarction by CT. *Am J Roentgenol* 1984; 143(5): 985–986.
10. Hilfiker PR, Weishaupt D, Marincek B. Multislice spiral computed tomography of subacute myocardial infarction. *Circulation* 2001; 104(9): 1083.
11. Klocke FJ, Simonetti OP, Judd RM, et al. Limits of detection of regional differences in vasodilated flow in viable myocardium by first-pass magnetic resonance perfusion imaging. *Circulation* 2001; 104(20): 2412–2416.

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