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Case Report

# Functional and metabolic improvement after coronary intervention for non-viable myocardium detected by $^{18}$F fluorodeoxyglucose positron emission tomography

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ABSTRACT

We report a case of a 64-year-old man suspected of myocardial infarction two months previously. Coronary angiography revealed total occlusion of the left anterior descending (LAD), and left ventriculography (LVG) showed remarkably reduced cardiac function and anterior dyskinesis. Electrocardiogram-gated thallium-201 Single Photon Emission Tomography (TL-SPECT) and $^{18}$F fluorodeoxyglucose positron emission tomography (FDG) were performed separately, and revealed large anterior myocardial infarction with markedly reduced tracer uptake, suggestive of non-viable myocardium. Percutaneous coronary intervention (PCI) was performed and stent was implanted successfully. Six months after PCI, LVG showed remarkable recovery in global function. Significant wall motion improvement and recovered glucose metabolism were observed in the infarcted myocardium despite having previously been diagnosed as lacking viability.

<**Learning Objectives:** In patients with left ventricular dysfunction, revascularized myocardium can contribute to improve cardiac function and prognosis. This evidence was established for old, or chronic status of myocardial infarction which is defined as over one month from onset of acute myocardial infarction. In this case report, we suggest that it can be premature to determine myocardial viability using FDG for the patient with under pre-chronic status after myocardial infarction due to underestimation on myocardial FDG uptake.>



## Introduction

Fluorodeoxyglucose (FDG)-positron emission tomography (PET) is widely known as a useful modality to identify viable myocardium compared to conventional radionuclide perfusion tracer [1,2]. It is recognized that viability testing can be useful to improve the prognosis of appropriate candidates. Generally, old or chronic myocardial infarction is defined as the status at least one month after the onset of acute myocardial infarction. Here we experienced a patient with chronic status of myocardial infarction who showed significant functional and metabolic recovery by percutaneous coronary intervention (PCI).

## Case report

A 64-year-old man was transferred to our facility with diagnosis of chronic status after myocardial infarction (presumed at least two months after onset). At the time of admission, he did not complain of any symptoms, and the detailed information about the myocardial infarction such as maximum creatinine kinase and duration of agina were unknown. He had multiple risk factors including hypertension, diabetes (HbA1c = 7.1%), and ex-smoker. Electrocardiogram (ECG) showed poor R wave progression in leads

*Abbreviations:* SPECT, Single Photon Emission Computed Tomography; FDG, $^{18}$F fluorodeoxyglucose; PET, Positron Emission Tomography; TL, thallium-201; QGS, Quantitative Gated Single Photon Emission Tomography; PCI, Percutaneous Coronary Intervention; Tc-99m, Technetium-99m.

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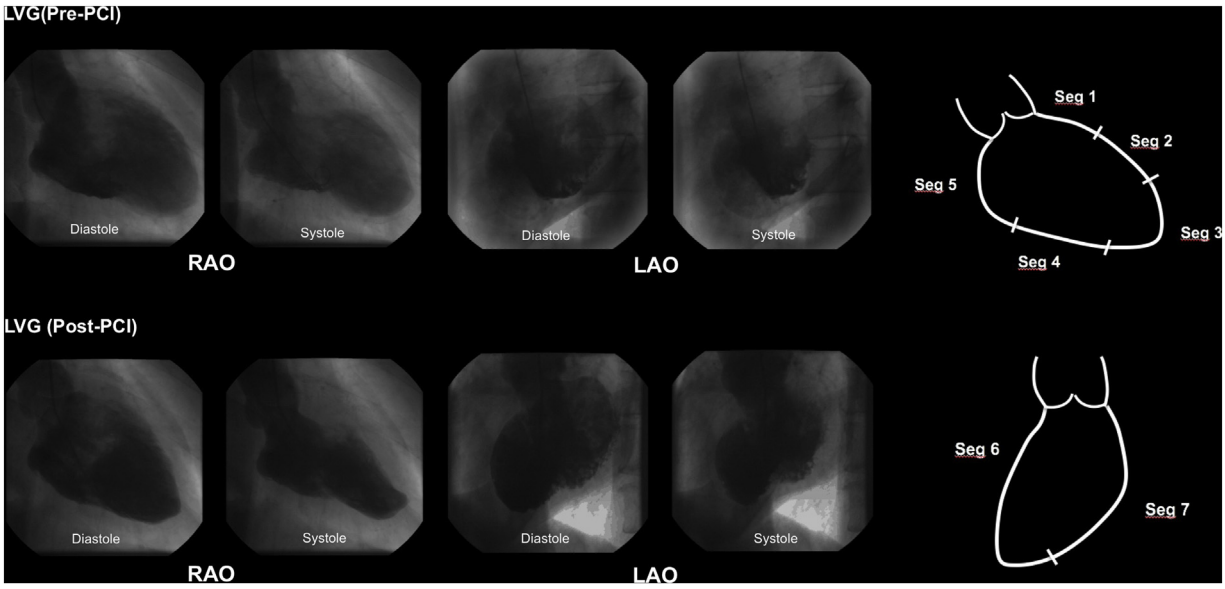


**Fig. 1.** Pre- and post-PCI left ventriculography (LVG) are shown. Diagrammatic presentation of right and left anterior oblique (RAO and LAO) views of the left ventricle segmentation (right). Pre-PCI LVG showed severely reduced global function and enlarged left ventricle (ejection fraction [EF]: 31%, End-diastolic volume [EDV]: 200 ml). Severe hypokinesis of anterolateral (seg 2) and dyskinesis of apical (segt 3) wall motion were found. Six months after PCI, LVG showed remarkably improved wall motion, and global cardiac function (EF: 44%, EDV: 177 ml).

V1-3 and Q wave in leads II, III, and aVF. Coronary angiography showed total occlusion of the left anterior descending artery (LAD, segment #6: 25%, #7: 100% with collateral flow from right ventricular branch, and diagonal branches arose from distal site), left circumflex artery (LCX, segment #13: 75%), and left ventriculography (LVG) demonstrated dilated left ventricle and severe hypokinesis of anterolateral (segment 2) and dyskinesis of apex (segment 3) (Fig. 1).

Adenosine stress/rest TL showed large anterior myocardial infarction with severe uptake reduction on rest images. Mild

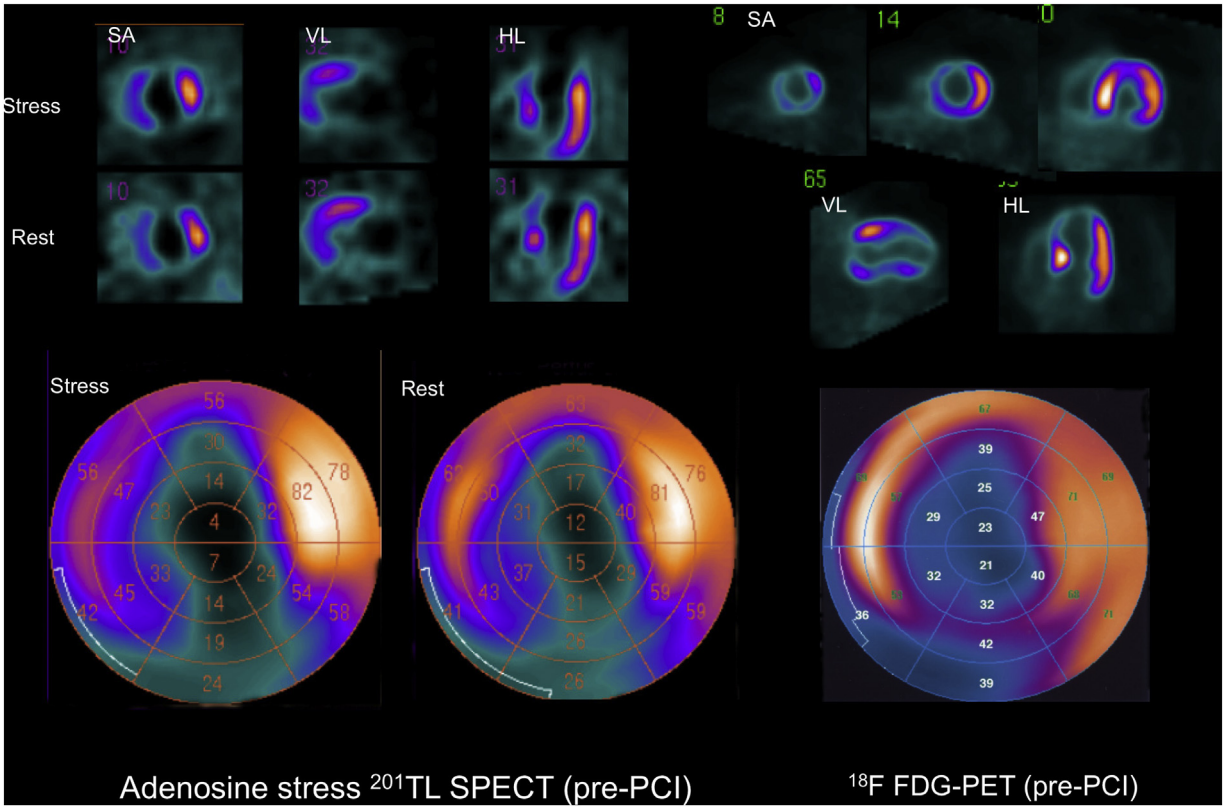


**Fig. 2.** Adenosine stress/ rest $^{201}$thallium perfusion SPECT (TL) and $^{18}$F FDG-PET (FDG) prior to PCI are shown (SA, short axis; VL, vertical long axis; HL, horizontal long axis). TL showed extensively wide sized defect in anterior to inferior area, and no transient ischemia was observed in infarct lesion. As well as TL, FDG showed global defect and %uptake of infarct area was lower than 50.

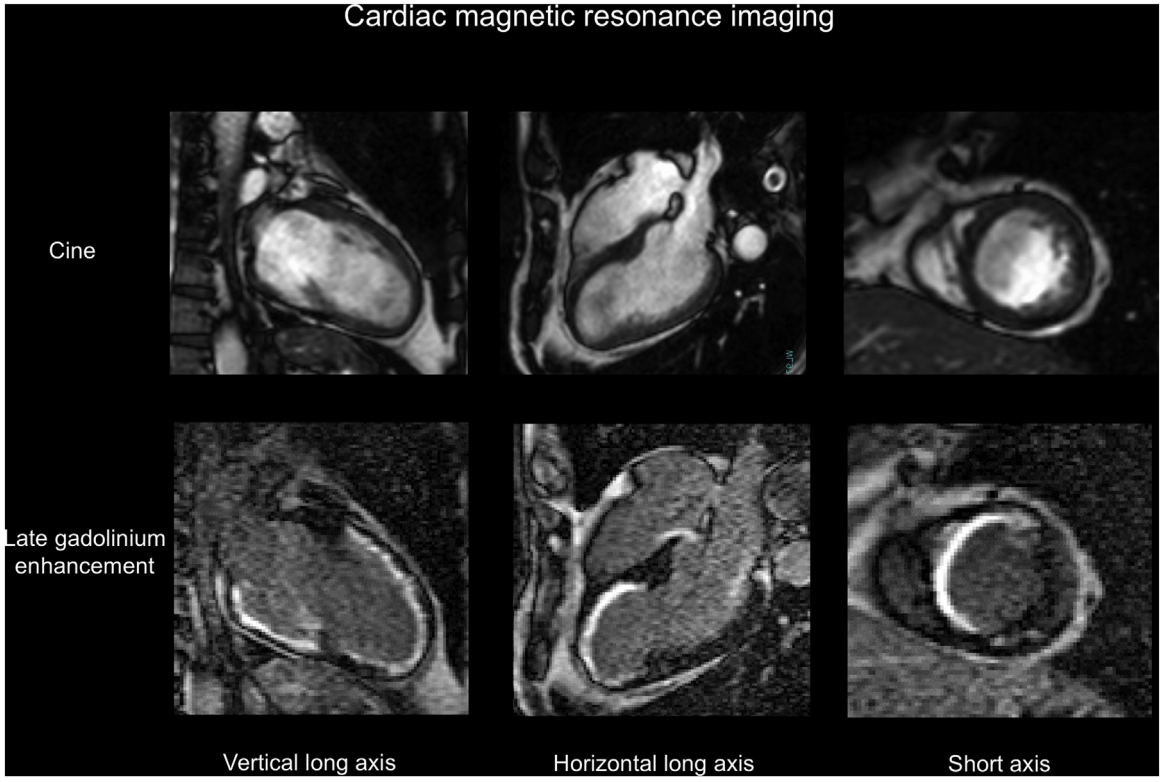


**Fig. 3.** Cardiac magnetic resonance imaging with late gadolinium enhancement (LGE) 3 months after PCI. Broad anterior and inferior infarction was observed. Viable rim with magnitude of LGE below 50% wall was detected in mid-anterior and apical-lateral area which indicated preserved myocardial viability.

ischemia was confirmed in septal area, but no transient ischemia was observed in lateral and infarct area (Fig. 2, left). For further evaluation of myocardial viability, ECG-gated F-18 FDG study was performed with a PET scanner (Siemens Accel, Hoffman Estates, IL, USA) according to standardized euglycemic-hyperinsulin clamp protocol (clamping interval: 1 h, acquisition starting 1 h after 185 MBq of FDG injection) [2]. The value of blood glucose concentration maintained around 100 mg/dL, and was 88 mg/dl at the time point of tracer injection. Wall motion and thickening was analyzed by Quantitative gated SPECT (QGS®, Cedars Sinai, Los Angels, CA.). In the FDG-PET study, severely low uptake was observed in anterior to infero-apical area and it was extensively less than 50% uptake, suggesting that the viability of the infarct area was unlikely. Visual score analysis by 4-point scale with American Heart Association 20-segment model showed summed defect score (SDS) as 50, and %total defect (%TPD) as 62.5 (over 40%). Accordingly, myocardial functional recovery through angioplasty was not expected (Fig. 2, right).

The patient had been scheduled for gastrointestinal operation for early stage malignant tumor. He was scheduled to undergo PCI (based on the assessment that ischemia was found in the septal wall, and needed to minimize the risk for cardiac events during operation) and a stent (Duraflex R $3.5 \times 25$ mm,Goodman®Nagoya,Japan) was successfully implanted to cover #6 distal to #7. The patient received medication during follow up (25 mg of atenolol, and 2.5 mg of enalapril). Three months after PCI, cardiac magnetic resonance (CMR) imaging exam was performed to determine cardiac contraction. As expected, significant late gadolinium enhancement in broad anterior and inferior area was observed. However, wall thickness was relatively preserved, and the viable rim (below 50% of myocardial wall) was found in mid-anterior, and apical-lateral (Fig. 3).

Follow-up coronary angiography was performed 6 months later and showed no in-stent restenosis or new obstructive vessel. LVG showed remarkable improvement in left ventricular (LV) function, compared with the previous study including anterior wall motion (end diastolic volume changes from 200 to 177 ml, end systolic volume from 137 to 76 ml and LV ejection fraction from 31% to 44%) (Fig. 1 lower). Follow-up FDG-PET study revealed that glucose metabolism significantly recovered in the revascularized myocardium, and SDS and %TPD improved to 41 and 51.3 (Fig. 4, upper). In comparison to TL and FDG pre PCI, mid-anterior (seg 7, 13) and apical-lateral (seg 17, 18) site of LV where myocardial viability had not been expected remarkably increased in FDG uptake (mid-anterior: from 39% and 25–59% and 46%; apical-lateral: from 47% and 49–69% and 52%), while apex, infero-basal, apical-inferior, and septal wall showed below 50%, namely remained non-viable. Interestingly, in addition to the recovery of myocardial metabolism, polar map and segmental analysis showed significant wall motion and thickening improvement (Fig. 4, middle and lower).

## Discussion

This case showed significant functional recovery after revascularization regardless of lack of myocardial viability diagnosed on FDG. FDG-PET is now widely thought as a standard and most reliable method for detecting myocardial viability with its high predictability [1–3]. Comparing other modalities such as magnetic resonance imaging and echocardiography, FDG-PET performs with high sensitivity, relatively low specificity, namely high negative predictive value. In a meta-analysis by Bax et al., FDG-PET showed sensitivity and specificity of 93% and 58%, respectively [4]. In usual assessment, less than 50% of myocardial uptake was considered to be non-viable. The degree of mismatch between myocardial perfusion and glucose metabolism was also thought to be a reliable factor to predict the functional recovery [5]. Likewise, Haas et al. reported that the functional recovery by revascularization is not to be expected when non-viable myocardium accounted for more

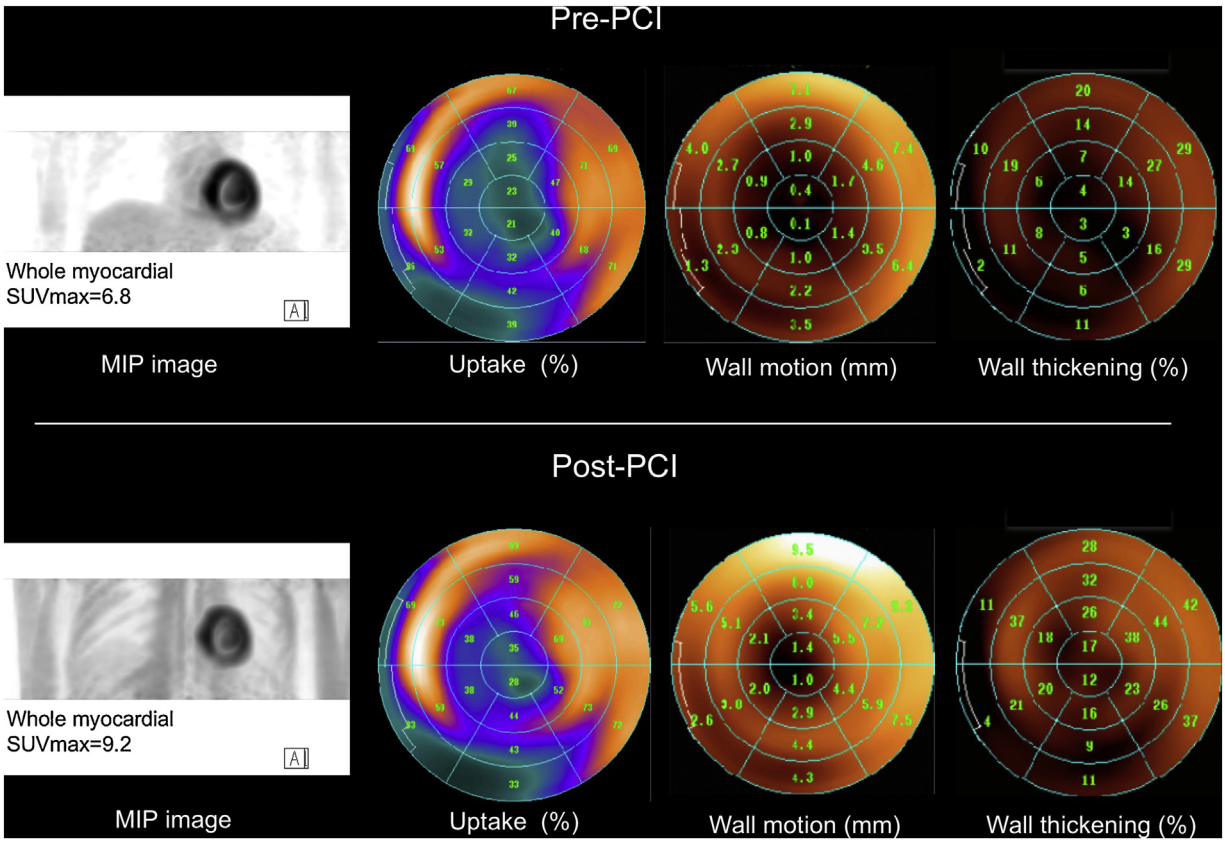


**Fig. 4.** Six months after PCI, significant reduction in size in FDG defect and improved % uptake were observed (top). Middle and lower show polar map analysis from ECG-gated FDG for pre- and post-PCI. Remarkable recovery of FDG uptake was observed in mid-anterior (seg 7, 13: from 39% and 25–59% and 46%), and apical-lateral (seg 17, 18: from 47% and 40–69% and 52%). The %uptake of apex, infero-basal, apical-inferior, and septal wall (seg 4, 10, 15–20) remained below 50%. Significant improvement in wall motion and thickness were found in the area where myocardial glucose metabolism has recovered by coronary intervention.

than 40% of whole heart [6]. Although these assessments were performed using N-13 ammonia for perfusion tracer, some investigators reported the usefulness of TL or Tc-99 m sestamibi as perfusion tracers combined with FDG to detect mismatch for viability testing [7,8]. In the current case, no perfusion-metabolism mismatch was observed, and defect size was more than 60% of whole myocardium. Despite these findings, the patient showed significant recovery in myocardial metabolism and kinetics. However, CMR after PCI showed viable rim in mid-anterior and apical-lateral wall while FDG uptake pre-PCI was lower than 50% in those areas. FDG-PET post PCI showed viable uptake in mid-anterior and apical-lateral wall which appeared to be a visually similar finding to CMR. Perrone-Filardi et al. reported that reverse perfusion-metabolism mismatch might occur in the patients with chronic ischemic heart disease [9]. These reports indicated that there were some cases which turned out to be underestimated in myocardial viability due to prolonged hypo FDG uptake, or might be in transition from acute stunning to hibernation. Prolonged myocardial stunning may cause the underestimation of viability in myocardial infarction. Although there might be some possible conditions which may affect diagnosis in FDG-PET such as partial volume effects, the outcome of our case suggests that reversible glucose hypometabolism may rarely exist in recent myocardial infarction. Metabolic restoration by interventional treatment might contribute to regional myocardial functional recovery.

## Acknowledgment

The authors have indicated they have no financial conflicts of interest.

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