Non-diagnostic ^{18}F-FDG PET myocardial viability studies in type-2 diabetic patients

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INTRODUCTION

^{18}F-FDG PET is a valuable functional imaging tool and the only FDA-approved technique for assessing viable myocardium in ischemic cardiomyopathy. It is based on the recognition that resting LV dysfunction may be reversible from myocardial hibernation/stunning, and not necessarily due to an irreversible myocardial scar.[1] We report a case highlighting the pitfall of ^{18}F-FDG PET myocardial viability study performed with the standardized protocol of oral glucose loading and intravenous regular insulin injections in a type-2 diabetic patient with non-diagnostic images.

CASE SUMMARY

A 73-year-old man with type-2 diabetes mellitus, hyperlipidemia, coronary artery disease (CAD), post-coronary artery by-pass graft, and ischemic cardiomyopathy (left ventricular ejection fraction = 34%) was referred for ^{18}F-FDG PET/CT myocardial viability testing. Stress and rest ^{99m}Tc-sestamibi SPECT myocardial perfusion imaging showed a severe perfusion defect in the lateral and inferior myocardium with minimal reversibility, consistent with large scar and minimal ischemia in the left circumflex and right coronary distributions (Figure 1).

Patient preparation for ^{18}F-FDG myocardial viability testing included overnight fasting, followed by oral glucose loading (25 g of TRUTOL®) and intravenous regular insulin injection as per a standardized protocol.[1] Fasting blood sugar was 193 mg/dL. Based on blood sugar levels post-oral TRUTOL® ingestion a total of 8 units of regular insulin were administered before ^{18}F-FDG injection. Blood glucose level at the time of ^{18}F-FDG injection was 135 mg/dL and imaging began at 83 minutes after tracer injection. Acquired images showed poor myocardial FDG uptake and, thus, were non-diagnostic (Figure 2).

DISCUSSION

The most common standardized patient preparation technique for myocardial viability evaluation with ^{18}F-FDG involves fasting for at least 6 hours, followed by oral glucose (25 to 50 g) loading and supplemental administration of intravenous regular insulin based on blood glucose levels.[1] ^{18}F-FDG PET imaging in type-2 diabetics can be challenging as they may have insulin resistance and/or production deficits leading to poor FDG uptake in the myocardium. Cellular metabolic changes in diabetic cardiomyopathy that can explain these findings include decreased glucose uptake, shift in substrate utilization with increased uptake of free fatty acids, decreased insulin signaling, and activation of transcriptional pathways.[2]

Poor myocardial FDG uptake in diabetics has been described previously by Vitale and colleagues from the University of Ottawa.[3] They evaluated image quality in 10 non-insulin dependent diabetic patients with CAD and severe LV dysfunction, who underwent 3 consecutive ^{18}F-FDG PET myocardial viability studies using different preparation protocols (including the standard protocol of oral glucose load and IV regular insulin boluses, hyperinsulinemic euglycemic clamp, and

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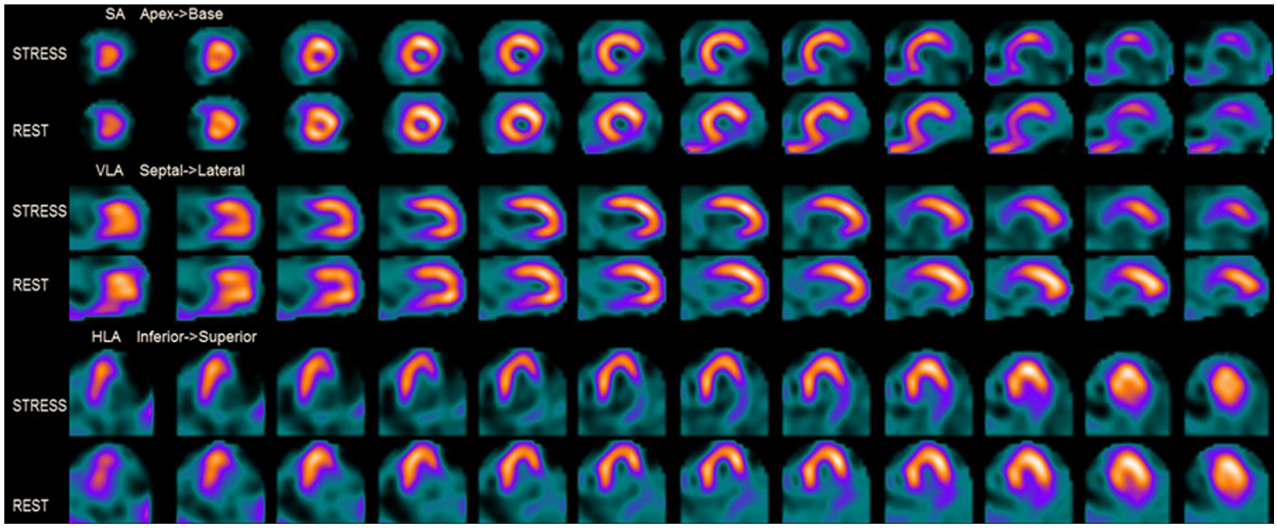

Figure 1. Stress and rest ^{99m}Tc-sestamibi SPECT MPI showing a severe large perfusion defect in the lateral and inferior myocardium with minimal reversibility, consistent with large scar and small ischemia in the left circumflex and right coronary distributions.

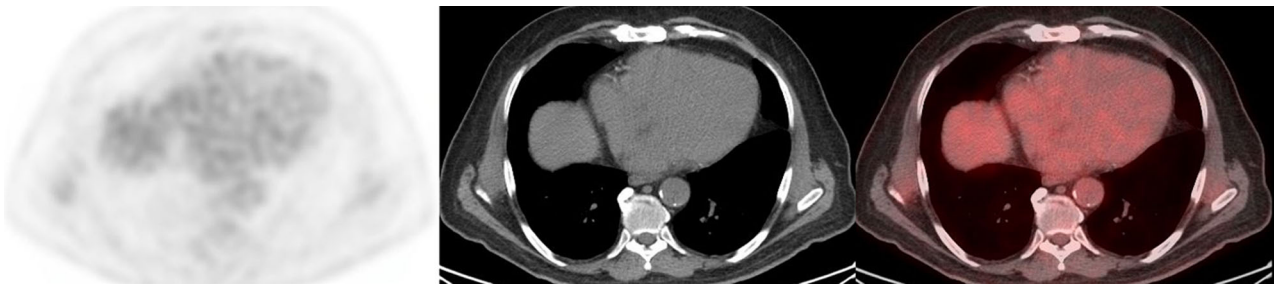

Figure 2. ^{18}F-FDG PET/CT viability study showing non-diagnostic images with myocardial FDG uptake similar to blood pool.

niacin). They found that 4/10 (40%) studies performed using the standard preparation protocol had poor image quality, in contrast to 2/10 (20%) studies prepped with the rigorous and time-consuming hyperinsulinemic euglycemic clamp protocol. Thus, the insulin clamp protocol may be the preferred method for detecting myocardial viability in this difficult patient population.

CONCLUSIONS

Non-diagnostic ^{18}F-FDG PET myocardial viability imaging in type-2 diabetic cardiomyopathy is a real and distinct possibility, despite adherence to a standardized patient preparation protocol of fasting, oral glucose loading, and IV regular insulin. The findings are attributed to altered cardiomyocyte substrate utilization with reduced glucose metabolism and enhanced fatty acid metabolism.

Disclosure

There are no COI with this work.

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