History
The patient is a 71-year-old female who presented with chest pain. She has a history of hypertension and high cholesterol. The patient has neither diabetes nor a family history of heart disease. The patient underwent Tc99m Tetrofosmin myocardial perfusion study that revealed a large fixed perfusion defect in the anteroseptal wall and apex of the myocardium (Figure 1). The clinical question is whether this represents post MI scar or hibernating myocardium due to chronic ischemia. If there is a significant area of hibernating but viable myocardial tissue, revascularization will improve survival. An FDG-PET scan was requested to address this issue.

PET Findings
Decreased FDG uptake is shown in the anteroseptal wall and apex as compared to the rest of the myocardium. However, the FDG uptake in the anteroseptal wall and apex is disproportionately enhanced when compared to the myocardial perfusion defect. This perfusion-metabolism mismatch indicates viable myocardial tissue in the anteroseptal wall and apex (Figure 2).

How Did PET Help?
The FDG-PET scan helped to detect hibernating but viable tissue in the area with a fixed defect on the perfusion study. Revascularization would be attempted to improve patient’s survival.

Discussion
FDG-PET is the gold standard for the evaluation of myocardial viability. A scar is characterized by concordant reduction in perfusion and FDG uptake (perfusion-metabolism match). A perfusion-metabolism mismatch is highly predictive of myocardial viability and indicates a high likelihood of improvement of cardiac function following revascularization. Studies have shown that cardiac morbidity and mortality are increased in patients with perfusion-metabolism mismatch. Studies have also shown that mortality ranged between 4 to 12% in the group of patients with matched defects, and between 33 and 41% in the mismatch group. In the mismatched group, if revascularization was performed, mortality dropped to between 4 and 12%.


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