Scintigraphic outlook of patients and regions with myocardial necrosis at myocardial perfusion scintigraphy

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Nudi et al. in their work entitled *Assessment of the Fate of Myocardial Necrosis by Serial Myocardial Perfusion Imaging*,¹ performed a retrospective analysis of SPECT (single-photon emission computed tomography) MPI (myocardial perfusion imaging) studies, where patients with repeat MPIs were included for indications other than acute coronary syndrome. Patients who underwent revascularization that was not performed in a setting of acute coronary syndrome were also included and further divided by LAD (left anterior descending coronary artery) and non-LAD perfusion territory revascularization. A novel necrosis score was used and the assigned score in each region was compared between the initial and repeat MPI study. MPI analysis was performed by assigning the highest degree of necrosis score to the patient. In the 3691 subjects, there were 25,837 segments, and 13% had necrosis. Only 48.9% of those with a clinical history of infarction had MPI evidence. Necrosis was highly reproducible on repeat MPI. There was no impact of either the site or type of revascularization in predicting necrosis on repeat MPI.

This is a very important work with a substantial number of patients included. The identification and quantification of myocardial necrosis/fibrosis in patients

with myocardial infarction (MI) is indeed a very important factor. The amount of infarcted myocardium plays an integral role in the evaluation of myocardial viability, where the amount of viability determines whether revascularization therapy can be of benefit and lead to potential regional and global myocardial functional recovery.

Myocardial infarction signifies irreversible ischemic damage to the myocardium leading to cardiomyocyte death. Due to very insignificant regenerative properties of the adult heart, myocardial necrosis leads to a scar formation. In 2012, the Joint Task Force of the European Society of Cardiology, American College of Cardiology Foundation, the American Heart Association, and the World Heart Federation (ESC/ACCF/AHA/WHF) released a third definition of acute MI. MI is defined as a clinical event consequent to the death of cardiac myocytes (myocardial necrosis) caused by ischemia (unlike non-ischemic etiologies such as myocarditis or trauma).^{2,3} Diagnosis of MI is based on upon a combination of the rise and/or fall of cardiac biomarker values (typically cardiac Troponin) with at least one value above the 99th percentile upper reference limit, and at least one of the following:

1. Symptoms suggestive of ischemia
2. New pathologic Q waves in the EKG
3. New significant ST segment—T wave EKG changes or new left bundle branch block
4. Presence of coronary thrombus by angiography or autopsy
5. New regional wall motion abnormality or loss of viable myocardium demonstrated by imaging
6. Cardiac death with symptoms suggestive of myocardial ischemia and presumed new ischemia ECG changes or new LBBB, but death occurred before cardiac biomarkers were obtained, or before cardiac biomarker values would be increased

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Nudi et al. in their work stated that “some degree of necrosis in MI is common.” This statement is certainly obvious by definition; it is the presence of necrosis, no matter how small or large, that makes the diagnosis of MI. However, it is true that various imaging modalities demonstrate certain limitations in their ability to identify acute or chronic MI, and furthermore may not be able to visualize small areas of necrosis. The diagnosis of myocardial infarction is done primarily clinically and by biomarkers. A group of patients in their study was diagnosed with MI based on a perfusion defect seen on myocardial perfusion imaging. While imaging plays a very important role in MI, it comes as an adjunct tool in making its diagnosis. Fixed perfusion defects on MPI studies are not specific to MI caused by atherosclerotic coronary artery disease. Such patterns can be seen with other scenarios, including non-ischemic myocardial injury such as myocarditis, trauma, sarcoidosis, infiltrative myocardial disease, and iatrogenic scarring. Soft tissue attenuation artifacts and anatomical variant of natural apical thinning can also be the culprits of fixed MPI defects.

MI size estimation has been validated by numerous trials and used as a marker of mortality endpoint in a setting of acute MI. One of the first works that evaluated criteria for myocardial viability against histopathological findings using Tc-99m Sestamibi was described by Dr. Verani et al.⁴ The authors demonstrated a close correlation between 99mTc-sestamibi activity and the extent of histologically documented myocardial viability in patients referred for CABG. Because a preserved cellular structure is the ultimate proof of viability, their results offered support for the use of 99mTc-sestamibi as a viability marker. There is a plethora of studies that attest to the fact of fixed perfusion defects related to MI decrease following revascularization or medical therapy.^{5,6} The study from Nudi et al. did not confirm that evidence and in part it may be lacking due to the exclusion of acute MI patients. Other explanations may be related to the absence of a correlation between the specific area of fixed perfusion defect and a target for revascularization. The authors did not offer the details concerning correlation between fixed defects and revascularized vessels with the exception of LAD versus non-LAD revascularization. It is possible that revascularization was performed in territories other than the true infarction area. Also, as stated above, some of the fixed perfusion defects identified on MPIs may not be related

to coronary artery disease but occurred due to non-ischemic etiologies, which are unlikely to yield any improvement following revascularization.

Lower administered activity of Tc-99m Sestamibi and Tetrofosmin (8–12 mCi) has been described to limit the ability to accurately assess MI size due to soft tissue attenuation effect in obese patients as well as increasing the image “noise”.⁷ Cardiac MRI has been considered the “gold standard” in the estimation of myocardial scarring volume and myocardial viability evaluation. Contrast MRI has demonstrated an excellent ability to distinguish between reversible and irreversible injury independent of wall motion and infarct age.⁸

The findings offered by Nudi et al. included a large patient database and can certainly be of benefit for future research endeavors. Correlation with other modalities offering higher spatial and temporal resolution can afford longitudinal evaluation of myocardial necrosis and offer interesting perspectives. The correlation between the myocardial infarct and left ventricular function will bring additional insight and value in patient management. Finally, the ability to reproducibly quantify myocardial infarction described by the authors can give more insight into determination of viability and treatment strategies efficacy.

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