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Episode 1

Prior to a nuclear cardiac myocardial perfusion exam, what are key patient preparation steps that improve the subsequent imaging?

Patients should be instructed to fast for something like 4 hours to reduce uptake in the GI system/liver. If possible, have patient refrain from taking certain medications to include long-acting nitrates, caffeine, calcium channel and beta blockers as these will reduce sensitivity for detection of a perfusion defect/coronary artery stenosis.

Is Tc-sestamibi or Tc-tetrofosmin more likely to show liver and bowel uptake on a cardiac imaging study?

Tc-tetrofosmin has more rapid liver and bowel clearance compared to sestamibi and therefore will have less competing /adjacent activity in the liver and bowel on a cardiac imaging study.

What is the purpose of adding physiologic or pharmacologic stress to a nuclear cardiac exam?

Stressing the patient with exercise (physiologic) or pharmacologic stress increases the sensitivity for detection of coronary artery stenosis. Without stress, nuclear cardiac imaging can detect something like stenosis >90%. With stress sensitivity improves to be able to detect a stenosis >50%.

Name some common pharmacologic vasodilators used for myocardial perfusion imaging?

Adenosine, dipyridamole and regadenoson are common pharmacologic vasodilators used for myocardial perfusion imaging.

What pharmacologic stress agent is a specific adenosine receptor agonist with a lower risk of inducing bronchospasm?

Regadenoson is a specific A2A adenosine receptor agonist with a lower risk of inducing bronchospasm compared to adenosine or dipyridamole. Note that adenosine typically is associated with the highest risk of side-effects, including a risk of AV block. Dipyridamole inhibits the breakdown of adenosine, thus raising adenosine levels to cause vasodilation.

Which agent has a longer half-life: regadenoson or adenosine?

Regadenoson has a half-life of 2-3 minutes and therefore may be given as a single IV bolus. Adenosine has a half-life of only 10 seconds and therefore is given as an IV drip.

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Which agents can be given to reverse the effects of regadenoson and other pharmacologic vasodilators?

Aminophylline is a reversal agent for regadenoson. Note that oral and IV caffeine also can provide reversal of the effects of regadenoson. Therefore, caffeine must be avoided prior to myocardial perfusion imaging when using any of the pharmacologic vasodilators in order to obtain adequate pharmacologic vasodilation/stress.

Which agent has a longer half-life: aminophylline or dipyridamole?

Aminophylline has a shorter half-life than dipyridamole. Therefore, when giving aminophylline to reverse the effects of dipyridamole, breakthrough symptoms can occur so you must continue to monitor for breakthrough when using aminophylline as a reversal agent.

Which pharmacologic agent increases stress by increasing heart rate and myocardial contraction as opposed to vasodilation?

Dobutamine is a beta 1 agonist that can be used to induce stress for myocardial perfusion imaging. This essentially causes a similar increase in heart rate and contractility to exercise instead of causing direct coronary vasodilation as with adenosine, dipyridamole and regadenoson.

Which patients may be best suited for pharmacologic stress with dobutamine?

Dobutamine may be considered for myocardial perfusion stress imaging in patients who cannot tolerate exercise for stress imaging who also have COPD and/or asthma to avoid the risk of bronchospasm that exists with the vasodilatory agents. Dobutamine can also be used in patients who have had caffeine intake within the past 12 hours. Note that dobutamine should not be used in patients on beta blockers as this would prevent the beta-1 induced increase in heart rate and contractility that is necessary to induce stress.

During a 1-day myocardial perfusion scan, is a higher dose of radiotracer administered during the stress or during the rest portion of the study?

Typically, you would start with a rest portion of the study at lower dose. Subsequently, a much higher dose would be given to overwhelm the rest counts and now provide perfusion information under stress. Note that a normal stress study effectively rules-out significant coronary artery stenosis, so if stress is performed first and is normal the rest portion of the study would not be necessary. 2-day protocols often would do stress first, and then rest the following day, only if stress is abnormal.

Basically, for a study with both stress and rest imaging, one either needs to wait about 4 half-lives for the radiotracer to decay from the first imaging study prior to performing the subsequent study to make sure there is not contamination on the second imaging from activity

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left over from the initial imaging. Alternatively, one can administer a dose something like 3-4 times as high for the second portion of the study and these will essentially overwhelm and diminish any residual counts that may be received from the initial imaging.

For more information on myocardial perfusion imaging, pharmacologic agents, patient contraindications for various agents, and protocols: please read here: https://www.asnc.org/files/Stress%20Protocols%20and%20Tracers%202009.pdf

Episode 2

By which mechanism does Thallium enter into a cell?

Thallium enters a cell using a Na/K pump in a manner related to blood flow.

What happens after Thallium enters into a cell?

Unlike Tc-sestamibi and Tc-tetrofosmin, Thallium is able to redistribute among viable cells in the myocardium. So initial Thallium imaging provides information about blood flow to the myocardium and delayed imaging evaluates for redistribution.

How does Thallium distinguish between normally perfused, ischemic, and dead (infarcted/scarred) myocardium?

Thallium can show areas of ischemic but viable myocardium. Ischemia: perfusion defect on initial imaging but uptake is seen on delayed imaging as Thallium redistributes into the viable but hibernating myocardium. Scar/prior infarct: perfusion defect on initial imaging that persists on delayed imaging (redistribution requires functional Na/K pumps—which won't be functioning in case of myocardial scarring). Normal perfusion: No defect on initial imaging. Delayed imaging would typically not be performed as ischemia and/or scar is already excluded if initial imaging is normal.

On a myocardial perfusion study with Tc-sestamibi or Tc-tetrofosmin, how does imaging distinguish between scar/prior infarct and ischemia?

The key to differentiate between scar and ischemia on a myocardial perfusion study is to compare uptake between the stress and rest portions of the examination. A fixed defect on both stress and rest is consistent with scar from prior infarct. A reversible defect, meaning a defect seen on stress imaging only, with no defect on rest imaging, is a manifestation of stress-induced ischemia. Sometime a fixed defect is seen with reversible components around the fixed defect (in other words the defect is distinctly larger on stress images and smaller on rest images) and this suggests a component of scar with peri-infarct ischemia.

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True or false: A thallium lung to heart ratio study with decreased lung uptake suggests multi-vessel coronary artery disease.

False. Multi-vessel coronary artery disease would show increased uptake in lungs as the reduced blood flow through the lungs due to cardiac disease is not sufficient to clear the tracer from the lungs.

What are imaging manifestations and clinical implications of transient ischemic dilation?

Transient ischemic dilation (TID) is identified on a myocardial perfusion scan when the left ventricular cavity appears larger on stress imaging compared to rest imaging. Note that perfusion can appear otherwise normal—the finding of interest is simply apparent left ventricular enlargement on stress imaging that is not seen on rest imaging. The significance of this finding is that this correlates with left main and/or 3-vessel coronary artery disease that causes diffuse subendocardial hypoperfusion. This diffuse subendocardial hypoperfusion means that less radiotracer is delivered to the subendocardial myocardium, therefore causing a diffuse subendocardial perfusion defect on stress imaging that causes the appearance of left ventricular enlargement on stress imaging, not seen on rest imaging (therefore transient dilation as in the name), thus showing diffuse reversible subendocardial ischemia related to left main and/or 3-vessel coronary artery disease.

How might dilated cardiomyopathy without ischemia manifest on a myocardial perfusion scan?

In comparison to transient ischemic dilation, dilated cardiomyopathy would be expected to present with left ventricular dilation that is fixed on both stress and rest images.

What is the significance of seeing increased right ventricular activity (right ventricle uptake similar to left ventricle uptake) on rest imaging?

If the right ventricle shows higher than normal uptake on rest imaging which would manifest as right ventricle uptake similar to left ventricle uptake this is a sign of right ventricular hypertrophy.

What is the difference between stunned and hibernating myocardium?

Stunned myocardium is seen in the acute phase following ischemia that resolves with reperfusion injury but not frank myocardial infarction/scarring and manifests as normal perfusion with impaired contractility of the heart. As the name suggests this stunning is temporary and typically resolves within a few weeks.

Hibernating myocardium results from chronic decreased perfusion to the heart from chronic severe coronary artery disease and manifests as areas of decreased perfusion and decreased contractility in myocardium that is not infarcted and is still viable. A key to remember for board

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exams is that hibernating myocardium is viable heart tissue that is chronically hypoperfused, and therefore poorly contractile but if the perfusion is improved through intervention such as coronary stenting or angioplasty, cardiac contractility can recover to normal or else be significantly improved.

How can one assess for viability of cardiac tissue using FDG PET/CT and/or Thallium?

Another way one could phrase this same question is as follows: how can one differentiate between infarcted myocardium and viable myocardium using FDG PET/CT and/or thallium imaging? With FDG-PET/CT hibernating myocardium is chronically hypoperfused and therefore very hungry for nutrients such as glucose—therefore FDG uptake is sometimes more intense in hibernating myocardium compared to normal myocardium. The key however is simply the finding of FDG uptake in the region of perfusion defect (whether increased, normal or slightly decreased compared to normal myocardium) proving that there are viable cells in the region of decreased perfusion/ischemia. With thallium imaging the key for viable myocardium is that thallium will redistribute into viable myocardium but not scarred myocardium. In hibernating myocardium on initial imaging with thallium one will see a perfusion defect related to the chronic hypoperfusion but on delayed imaging thallium will use functional NaK pumps to redistribute into any myocardium that is viable but will not redistribute into any myocardium that is scarred as scarred myocardium will not have functional NaK pumps.

Summary of imaging for:

Hibernating myocardium: myocardial perfusion scan will show decreased perfusion and decreased contractility, FDG PET/CT will show uptake equal to higher than normal for myocardium, thallium will show redistribution on delayed imaging. Hibernating myocardium is chronically hypoperfused myocardium that is still viable.

Stunned myocardium: myocardial perfusion imaging will show normal perfusion but decreased contractility. This is temporary and is the result of a recent, transient ischemic insult to the heart but not frank infarction.

Ischemic myocardium: myocardial perfusion scan will show less tracer uptake on stress compared to rest imaging (reversible defect) and poor contractility in ischemic segments on stress imaging.

Scar/infarcted myocardium: No tracer uptake on stress or rest images in the infarcted/scarred myocardium and no contractility in that infarcted segments as the myocardium is simply dead in that region. No redistribution with thallium and no uptake with FDG PET/CT.

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Episode 3

What is the primary purpose of a MUGA scan?

The primary purpose of a MUGA scan is to provide an ejection fraction calculation. MUGA stands for multi-gated acquisition scan and uses gating to provide evaluation of cardiac function and contractility after equilibration of radiotracer in the intravascular space.

What radiotracer is classically used for a MUGA exam?

MUGA classically uses a Tc-99m tagged red blood cell technique.

If you see a photopenic halo around the cardiac blood pool what does this suggest on a MUGA scan?

A photopenic halo around cardiac blood pool is a potential manifestation of a pericardial effusion.

If structures such as the pulmonary artery, thoracic aorta, left atrium, or right ventricle overlap the left ventricle on a MUGA acquisition will this falsely lower or falsely increase estimation of ejection fraction on a MUGA scan?

Vascular structures that overlap the left ventricle on a MUGA scan, whether great vessels or other cardiac chambers, will give the false appearance that tracer is not exiting the left ventricle and this will therefore lower the estimated ejection fraction. If you are asked this question, simply think it through and realize that in this scenario of vascular overlap, tracer will not be clearing the left ventricle as it should and this would therefore allow for underestimation of left ventricle ejection fraction.

If the background region of interest is erroneously placed over the spleen will this falsely lower or falsely increase estimation of ejection fraction on a MUGA scan?

Erroneously placing the background region of interest over the spleen will falsely increase ejection fraction calculation. The reason for this false increase in calculated ejection fraction is that there will be over-subtraction of background that is in the denominator of the ejection fraction equation, therefore this will lower the denominator and increase the calculated ejection fraction. The equation is (end diastolic counts – background) – (end systolic counts – background)/ (end diastolic counts – background).

Is tagging of Tc to red blood cells for a MUGA scan typically performed using in vivo or in vitro labeling?

For MUGA one can get by with in vivo labeling of red blood cells to Tc. This is different from a tagged red blood cell scan performed for a GI bleed which requires a higher labeling efficiency and requires in vitro labeling.

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What are some key features to identify the short axis, vertical long axis, and horizontal long axis on nuclear cardiac imaging?

Short axis: look for the round donuts or ring-like configuration of the left ventricle. Vertical long axis: to me looks like an arrow pointing to the right. The apex of the heart is at the right of the screen. Horizontal long axis: look for the two parallel vertical lines, similar to the two parallel long lines of an "H" (H=Horizontal).

What is the leading cause of attenuation artifact on nuclear cardiac imaging?

Breast tissue overlying the heart is likely the top cause of attenuation artifact.

What is the top cause of reconstruction artifact on a myocardial perfusion scan?

Bowel or liver uptake "stealing" counts from the heart causes reconstruction artifact.

What is the calculation for maximal heart rate used to determine whether somebody has reached sufficient cardiac stress for a Bruce treadmill protocol?

220 minus the patient's age. In general, it is desired to reach 85% of this maximal heart rate value for the stress portion of myocardial perfusion imaging.

What is the most commonly used nuclear medicine agent for evaluation of cardiac sarcoidosis?

FDG is the agent most commonly used in nuclear medicine to evaluate for cardiac sarcoidosis. On an FDG-PET/CT study active cardiac sarcoidosis shows heterogeneous myocardial uptake. Note that unlike amyloidosis and other infiltrative diseases of the heart, myocardial sarcoidosis is typically patchy and this patchy involvement accounts for the heterogeneous, slightly patchy uptake seen on a positive FDG-PET/CT study. A negative scan shows no increased myocardial uptake of FDG.

What patient preparation steps need to take place in order to prepare for FDG-PET/CT imaging for cardiac sarcoidosis?

Dietary modifications in which patients follow a high fat and low carbohydrate diet the day prior to imaging, with an overnight fast, is necessary. This switches myocardial metabolism away from glucose metabolism to free fatty acid metabolism. The purpose is to decrease the physiologic uptake of glucose in the heart with the rationale that areas of cardiac sarcoidosis will not switch their metabolism away from glucose uptake, thus allowing maximal signal to noise with areas of cardiac sarcoidosis taking up the FDG while areas of normal myocardium will take up less FDG as normal myocardium has preferentially switched to fatty acid metabolism. This is termed myocardial suppression and you need good myocardial suppression in order to get a high quality FDG-PET/CT scan for cardiac sarcoidosis. Some protocols also use IV heparin prior to imaging that can further suppress myocardial FDG uptake.

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True or false: patients being evaluated for cardiac sarcoidosis typically complete a myocardial perfusion scan in addition to an FDG-PET/CT study?

True. FDG images for inflammation related to sarcoidosis but this does not give information about perfusion of the heart. Comparing information regarding inflammation from FDG and perfusion from a nuclear perfusion agent such as Rubidium on PET or a Technetium agent allows differentiation of normal tissue from scar tissue or areas of active sarcoidosis. This is also important for staging as sarcoid progresses from normal (perfusion and FDG both normal) to early stage (mild perfusion defect, mild increased FDG uptake) and progressive stages wherein perfusion defects worsen, and FDG uptake progressively gets more pronounced. Finally, end stage fibrosis from sarcoidosis would show a severe perfusion defect with minimal to no FDG uptake.

What is the preferred agent for nuclear cardiac imaging of amyloidosis?

Tc 99m pyrophosphate (PYP) is the main agent used in the US for nuclear imaging of ATTR amyloidosis. Tc 99m pyrophosphate binds to deposited amyloid transthyretin protein (ATTR) in the myocardium.

What are key features of a positive Tc 99m pyrophosphate scan for ATTR cardiac amyloidosis?

An uptake ratio of 1.5 or higher of the heart to contralateral chest on a planar view is positive for ATTR deposition in the heart. Semi-quantitative measures are also used (0-absent cardiac uptake, 1 uptake less than bone, 2 uptake equal to bone, 3 uptake greater than bone) but for board purposes I think remembering the 1.5 cutoff of heart to contralateral lung uptake is the most important measure to remember.

See this great article for more information on cardiac sarcoidosis and nuclear imaging: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC4009625/