Spontaneous Silent Myocardial Ischemia Assessed by Technetium-99m-Sestamibi Imaging

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We report on an 80-yr-old patient with a history of inferior myocardial infarction. The patient was injected with 15 mCi of ^{99m}Tc-sestamibi when an electrocardiogram revealed new asymptomatic inferolateral ST-segment depressions. A second ^{99m}Tc study was performed 72 hr after the initial injection and after ST-segment changes had resolved. Scintigraphic acquisitions using SPECT revealed a large reduction in photon activity in the inferolateral and inferoposterior walls with 70%–80% normalization. Therefore, ^{99m}Tc-sestamibi imaging is useful in assessing asymptomatic silent myocardial ischemia.

J Nucl Med 1993; 34:134–136

Silent myocardial ischemia has been found in more than 50% of patients with unstable angina and is a marker of early unfavorable outcome (*1*). Diagnosis is made in the setting of electrocardiographic ischemic changes in the absence of accompanying symptoms. However, the extent and severity of silent ischemia is not accurately assessed by electrocardiography. Technetium-99m-sestamibi is a new perfusion imaging agent utilized in the evaluation of ischemic myocardium during exercise or pharmacological stress (*2*). Its use in the evaluation of chest pain during unstable angina has been previously described (*3*). In the present case, however, we report its use in a patient with spontaneous electrocardiographic changes in the absence of symptoms to determine the presence, location and severity of myocardial ischemia.

CASE REPORT

The patient was an 80-yr-old white female with a history of an inferior myocardial infarction 8 wk prior to evaluation. The patient subsequently presented with unstable angina, during which time there was no evidence of myocardial necrosis. She was scheduled for an outpatient dipyridamole ^{99m}Tc-sestamibi myocardial perfusion imaging study. The last episode of chest discomfort she had experienced was three days earlier. Pre-study vital signs and physical examination were unremarkable. However, a routine resting 12-lead electrocardiogram revealed new inferolateral ST-segment depressions compared with her baseline electrocardiogram of one month ago (Fig. 1). Pharmacologic infusion was therefore cancelled and the patient was immediately injected with 15 mCi of ^{99m}Tc-sestamibi and imaged 60 min later. She was admitted to the hospital, monitored, and serial cardiac enzymes were normal. Her electrocardiogram returned to baseline within the first 12 hr after admission (Fig. 1) and remained unchanged during her hospital stay. She remained asymptomatic during her admission. A second ^{99m}Tc-sestamibi study (15 mCi), again at rest, was performed 72 hr following the first with imaging 60 min after injection.

Scintigraphic acquisitions for both studies were performed with a large field of view ADAC ARC 4000 gamma camera using single-photon emission computed tomography (SPECT) and a low-energy, high-resolution, parallel-hole collimator. Sixty-four projections were acquired over a 180° arc on a 64 by 64 matrix. The initial images (Fig. 2) showed a large reduction in photon activity in the inferolateral and inferoposterior walls. The second rest study revealed 70%–80% normalization with a residual defect consistent with the history of prior inferior myocardial infarction. She was referred for cardiac catheterization which showed an 85% proximal right coronary artery stenosis and a total occlusion of the left circumflex artery prior to the first obtuse marginal branch which filled via left to left collaterals. Her left ventriculogram showed an infero-posterior hypokinesis. She underwent a successful angioplasty to her right coronary artery and is doing well.

DISCUSSION

This case illustrates documentation of spontaneous silent myocardial ischemia with a resting injection of ^{99m}Tc sestamibi. The study was instrumental in identifying the degree and location of the ischemia, allowing proper subsequent therapy. The finding of a perfusion defect in the absence of angina is consistent with the concept of the ischemic cascade (*4*). It has been suggested that ischemia begins with an imbalance between myocardial oxygen supply and demand followed by electrocardiographic changes and, in some cases, by the subsequent clinical appearance of ischemia, i.e., angina pectoris. Chierchia et al. have demonstrated that a decrease in coronary sinus blood oxygen saturation consistently precedes the onset of hemodynamic, electrocardiographic or symptomatic evidence of ischemia (*5*). In their study, only 16% of 293 episodes of ischemic electrocardiographic changes were

Received Jun. 3, 1992; revision accepted Aug. 6, 1992.

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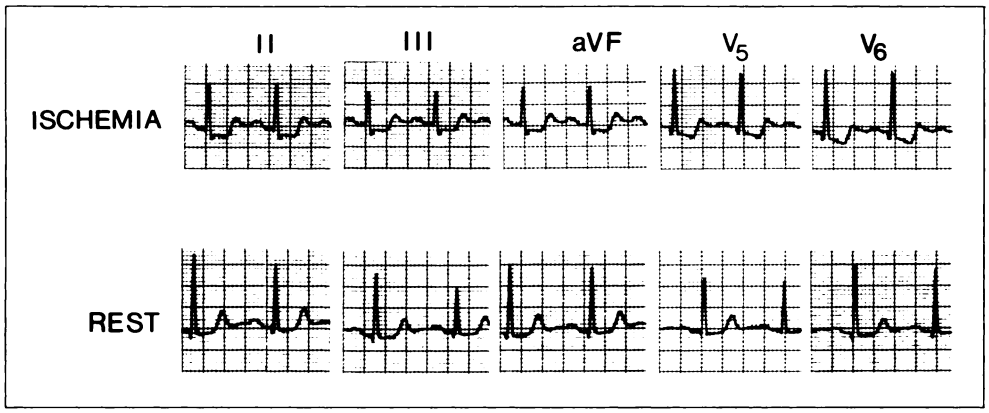

FIGURE 1. Surface electrocardiogram during spontaneous myocardial ischemia (ischemia) and at follow-up 72 hr later (rest). Marked ST-segment depressions on the initial tracings are resolved in the second study.

accompanied by angina, and when angina did occur it was typically preceded by electrocardiographic evidence of ischemia.

Nuclear imaging studies using ^{201}Tl initially and ^{99m}Tc-sestamibi more recently have been used by several investigators to evaluate unstable angina. Wackers et al. (*6*) found that an abnormal scintigraphic image from a single study during pain-free periods in patients with unstable angina was predictive of a more complicated course and did not necessarily imply myocardial infarction. Bilodeau et al. (*3*) reported on the usefulness and feasibility of ^{99m}Tc-sestamibi SPECT imaging for the diagnosis and prognosis of coronary artery disease in the setting of spontaneous chest pain with the diagnosis of unstable angina. The sensitivity of scintigraphy after the injection of ^{99m}Tc-sestamibi during chest pain was 96% and the specificity (79%), both of which were higher than clinical and electrocardiographic data.

The etiology of the defect seen during a pain-free state in patients with unstable angina may be attributed to an unrecognized prior myocardial infarction, an evolving myocardial infarction or the presence of spontaneous silent ischemia or chronic myocardial ischemia. None of the prior studies evaluating unstable angina patients imaged during pain-free states reported on the electrocardiographic changes during imaging or after resolution of symptoms. In the case reported, the use of ^{99m}Tc-sestamibi imaging allowed documentation of the extent and location of ischemia without subjecting the patient to a potentially increased risk of pharmacological or exercise stress testing in the setting of unstable angina. The use of ^{99m}Tc-sestamibi offers a potential time advantage over ^{201}Tl since ^{99m}Tc-sestamibi is not substantially redistributed. Thus, patients may be injected with the radiopharmaceutical in the unstable period, stabilization of the patient can be accomplished and imaging performed up to 6 hr later. Likewise, reinjection and reimaging can be accomplished when the patient is stabilized. Thus, ^{99m}Tc-sestamibi imaging is a useful modality in evaluating patients with spontaneous silent myocardial ischemia.

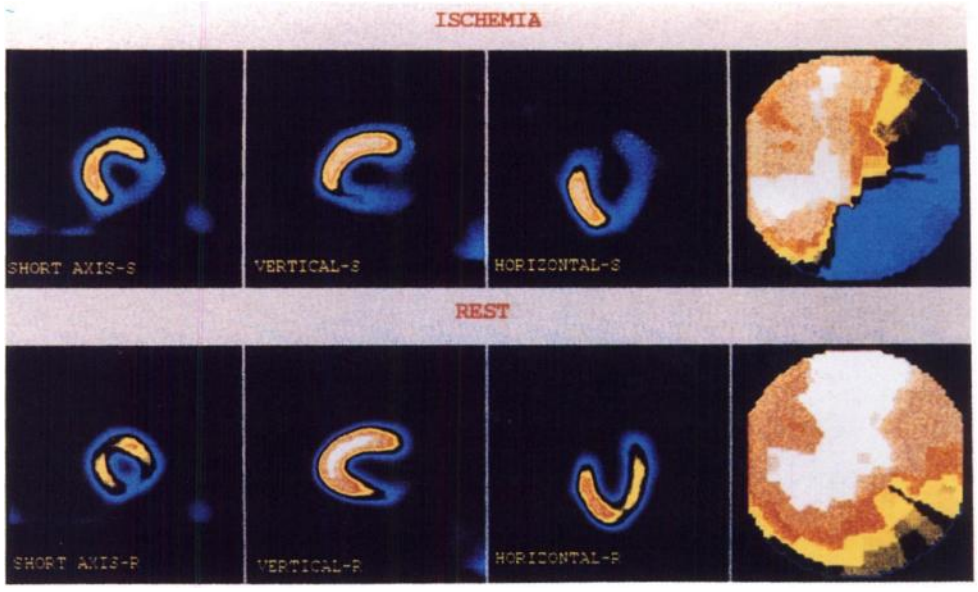

FIGURE 2. Technetium-99m-sestamibi SPECT images obtained during spontaneous myocardial ischemia (ischemia) and at rest (rest) 72 hr later. Defects were seen in the inferolateral and inferoposterior walls with normalization on the rest images and a residual inferior defect. Bullseye reconstruction confirms an inferolateral defect with marked improvement upon rest imaging.

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