

Reverse Redistribution on Dynamic Exercise and Dipyridamole Stress Technetium-99m-MIBI Myocardial SPECT

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Reverse redistribution on ^{201}Tl -chloride stress-redistribution myocardial scintigraphy has been associated with coronary artery stenosis. We report a patient whose two separate $^{99\text{m}}\text{Tc}$ -MIBI myocardial SPECT stress studies (dynamic exercise and dipyridamole) showed septal reverse redistribution and fixed inferior defect. Echocardiograms showed left ventricular (LV) hypertrophy and diffuse hypokinesis, especially in the inferior wall, and EKG showed LV hypertrophy and strain and inferior infarct. Coronary angiogram confirmed two-vessel disease involving 80%–90% stenosis of the proximal second diagonal branch of the left anterior descending artery and 75%–90% stenosis of the right coronary artery as well as global left ventricular dysfunction. Reverse redistribution on $^{99\text{m}}\text{Tc}$ -MIBI myocardial SPECT occurring on dynamic or dipyridamole stress may indicate damaged but viable myocardium.

Key Words: reverse redistribution; technetium-99m-MIBI; dipyridamole stress testing; single-photon emission computed tomography

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Reverse redistribution on ^{201}Tl -chloride myocardial SPECT is defined as normal uptake on stress becoming decreased uptake on rest imaging (1–7). This phenomenon occurred in 5%–21% of exercise ^{201}Tl studies (2–6). The clinical significance and pathophysiological mechanisms of reverse redistribution on exercise ^{201}Tl scintigraphy in patients with coronary artery disease (CAD) are debatable: Some studies have associated reverse redistribution with presence of significant CAD (1,2,6,7), while others have not (3–5). Reverse redistribution has also been related to the presence of scar and normal tissue (2,6) or to patients with cardiac event after thrombolytic therapy for acute infarction (6,8).

Myocardial tissue of the reverse redistribution region is subject to ischemia or hypoxia that does not produce cell death (9). Soufer et al. indicated that 72% of segments with

reverse redistribution on planar ^{201}Tl scintigraphy were viable by PET (10). It has been theorized that reverse redistribution occurs when damaged myocardium shows initial normal ^{201}Tl uptake and rapid ^{201}Tl washout (9–11). Reverse redistribution may occur after exercise (1–11) or dipyridamole stress (12). We report a patient who had two dynamic $^{99\text{m}}\text{Tc}$ -MIBI SPECT scans (one exercise and one dipyridamole stress) that showed reverse redistribution of the septal wall that proved to be severe CAD with global left ventricular (LV) dysfunction.

CASE REPORT

A 60-yr-old man, who had suffered with chest pain for 1 yr, had a history of hypertension and congestive heart failure believed to be diastolic dysfunction. On examination, he was in no acute distress with blood pressure 160/100 and had distant with normal S1 and S2 heart sound. A few wheezes at the lung bases were noted as well as bilateral pretibial pitting edema. An echocardiogram showed diffuse hypokinesis, especially in the inferior wall, and LV hypertrophy. Chest radiograph demonstrated cardiomegaly.

The patient underwent a 2-day protocol of $^{99\text{m}}\text{Tc}$ -MIBI rest and dipyridamole stress SPECT imaging using a three-head gamma camera. The patient received 59.6 mg dipyridamole intravenously and then 30 mCi $^{99\text{m}}\text{Tc}$ -MIBI intravenously. The patient was imaged approximately 1.5 hr after tracer injections after drinking 8 ounces whole milk 30 min postinjection to facilitate gallbladder emptying. Images were acquired in a 64×64 matrix in the step-and-shoot mode, with 40 steps of 3° each for 15 sec per step at 1.3 magnification. Total acquisition time was 14.7 min for each phase. Data were then prefiltered with a low-pass order of 4.0, cutoff 0.25, and were reconstructed 360° with a ramp filter. Technetium-99m-MIBI myocardial SPECT showed decreased uptake in the inferior wall and normal uptake in the rest of the left ventricular wall. Rest $^{99\text{m}}\text{Tc}$ -MIBI SPECT images obtained the next day showed decreased uptake in the inferior and septal walls, consistent with a fixed defect of the inferior wall and reverse redistribution of the inferoseptal wall (Fig. 1). Inferoseptal reverse redistribution was demonstrated on a previous dynamic stress-redistribution $^{99\text{m}}\text{Tc}$ -MIBI SPECT scan (Fig. 2) obtained 6 mo earlier; three echocardiograms were also obtained at this time, which showed diffuse hypokinesis, especially in the inferior wall.

Concurrent cardiac catheterization revealed two-vessel coronary disease: 80%–90% stenosis in the proximal second diagonal branch of the left anterior descending (LAD) artery, with luminal irregularities throughout the remainder of the LAD system and

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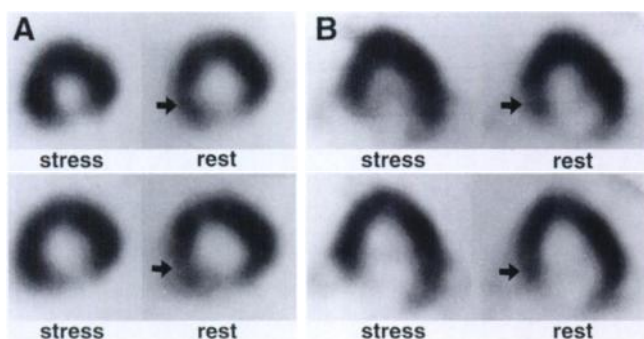


FIGURE 1. (A) Technetium-99m-MIBI myocardial SPECT (short-axis) shows decreased uptake in the inferior wall and normal uptake in the remainder of the LV wall on stress imaging and decreased uptake in the inferior and infero-septal walls (arrow) on rest imaging. (B) Technetium-99m-MIBI myocardial SPECT (horizontal long-axis) shows decreased uptake in the infero-septal wall (arrow) on imaging rest and normal uptake of the infero-septal wall during stress phase.

75%–90% stenosis at the crux of the right coronary artery (RCA) prior to origin of posterior descending artery (Fig. 3). Contrast left ventriculography demonstrated global hypokinesis (which was worse in the inferior wall), with an ejection fraction of 35% ($n \geq 50\%$). Four months after rest and dipyridamole ^{99m}Tc -MIBI SPECT imaging, repeat echocardiography again demonstrated diffuse hypokinesis, especially in the inferior wall. The patient's EKG on the day of the first ^{99m}Tc -MIBI myocardial SPECT scan was normal sinus with LV hypertrophy. The EKG from 3 wk before the second ^{99m}Tc -MIBI SPECT and thereafter showed LV hypertrophy and strain and a previous inferior transmural infarct.

DISCUSSION

We used ^{99m}Tc -MIBI to demonstrate reverse redistribution during dipyridamole stress and dynamic exercise myocardial SPECT. Diffuse hypokinesis was consistent, especially in the inferior wall on the four echocardiograms. Persistent worsening inferior hypokinesis on the echocardiograms, along with fixed inferior hypoperfusion on the MIBI images, might account for the EKG findings of inferior infarct.

Significant stenosis of the RCA and second proximal branch of the LAD may contribute to diffuse hypokinesis; worsening inferior hypokinesis may reflect fixed inferior

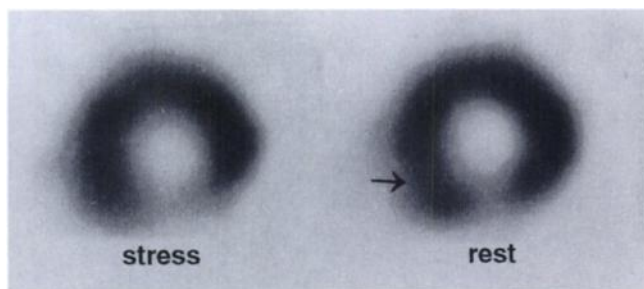


FIGURE 2. Stress-redistribution ^{99m}Tc -MIBI myocardial SPECT (short-axis) performed 6 mo prior to Figure 1 images show relatively decreased uptake in the inferior septal wall (arrow) during redistribution and normal uptake during stress (reverse redistribution), fixed inferior hypoperfusion and presence of transient LV dilatation.

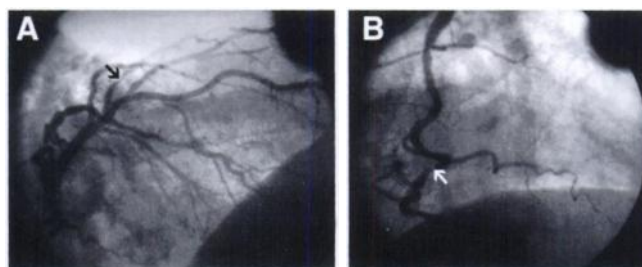


FIGURE 3. (A) Left coronary angiography shows 80%–90% stenosis (arrow) in the second diagonal branch proximal to the LAD with luminal irregularities throughout the remainder of the LAD. (B) Right coronary angiography shows 75%–90% stenosis (arrow) at the crux of the RCA prior to origin of the posterior descending artery.

hypoperfusion, apparently due to inferior infarct. Coronary angiographic demonstration of significant stenosis of the RCA and second proximal diagonal LAD provided anatomical information, whereas MIBI imaging provided myocardial functional status: inferior fixed defect, infarct (significant RCA stenosis) and reverse redistribution, damaged but viable myocardium (also significant stenosis of the proximal second diagonal stenosis of the LAD). Technetium-99m-MIBI is taken up in the myocardium as a function of blood flow. Myocardial uptake is slow because of a mechanism that depends on concentration and the potential gradient across the cellular membrane and mitochondria, as well as prolonged retention in the myocardium with negligible redistribution. MIBI requires administering two injections on routine stress-redistribution images. The cause of rapid washout rates in damaged myocardium in reverse redistribution ^{201}Tl myocardial SPECT (6,9–11) could not be explained on reverse redistribution ^{99m}Tc -MIBI myocardial SPECT images.

Reverse redistribution in chronic CAD reflects viable myocardium that critically depends on collateral circulation (1). Dilatation of collateral circulation induced by dipyridamole stress or dynamic exercise, which then supplies damaged yet viable myocardium, may explain our patient's reverse redistribution on ^{99m}Tc -MIBI SPECT.

CONCLUSION

Reverse redistribution may be demonstrated on ^{99m}Tc -MIBI myocardial SPECT after dynamic or dipyridamole stress and may indicate damaged myocardium with LV systolic dysfunction.

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