Technetium 99m Stannous Pyrophosphate Myocardial Imaging in Patients with and without Left Ventricular Aneurysm

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SUMMARY To further explore the usefulness of technetium 99m pyrophosphate (99mTc-PYP) myocardial imaging and test its validity in the diagnosis of acute myocardial infarction, 99mTc-PYP myocardial scintigrams were performed in 50 patients. Out of 28 patients with acute myocardial infarction, myocardial scintigrams demonstrated localized activity in the 15 patients with transmural, and diffuse activity in the 13 patients with subendocardial myocardial infarction. Twenty-two patients with significant coronary artery disease documented by coronary angiography but without acute myocardial infarction were also studied. Nine of ten patients with clinical evidence of left ventricular aneurysm from previous myocardial infarction and definite left ventricular dyskinesia had positive scintigrams with activity localized to the site of the wall motion abnormality. Two of five patients without definite aneurysm but with left ventricular akinesis also had localized uptake in the involved area of the left ventricle. Seven patients with normal left ventricular wall motion had negative scintigrams. These findings suggest caution in interpreting positive 99mTc-PYP scintigrams as being indicative of acute myocardial infarction when evidence of a left ventricular aneurysm is also present.

MYOCARDIAL IMAGING with 99mTc-PYP has been recently reported to be a reliable noninvasive technique in the diagnosis of acute myocardial infarction.1, 2 Experimental infarcts are known to display scintigraphic activity approximately 12 to 16 hours after infarction with progressively decreasing activity over the next one to two weeks.2 Positive myocardial scintigrams with localized activity are associated with appropriate cardiac enzyme and/or electrocardiographic findings of acute transmural myocardial infarction.1 Some patients with unstable angina pectoris and occasional patients with other types of chest pain have been reported to demonstrate positive scintigrams in the absence of conclusive electrocardiographic or enzymatic evidence of acute myocardial infarction.3

This communication reports our experience with 99mTc-PYP myocardial scintigrams in patients with acute myocardial infarction and in patients with previous myocardial infarction and documented ventricular aneurysm or other wall motion abnormalities without evidence of acute myocardial infarction.

Methods

99mTc-PYP myocardial scintigrams were performed in 50 patients. Twenty-eight patients had acute myocardial infarction, and 22 had significant coronary artery disease documented by coronary angiography without acute myocardial infarction. After informed consent had been obtained, myocardial scintigrams were performed in the Nuclear Medicine Laboratory. Patients with acute myocardial infarction were studied under constant electrocardiographic monitoring with emergency drugs and a defibrillator immediately available. Myocardial imaging was performed in anterior, lateral, and left anterior oblique positions 45–60 minutes after the intravenous injection of 15 mCi of 99mTc-PYP tagged to 5 mg of stannous pyrophosphate. Average duration of imaging in each patient was 20 minutes. Repeat scintigrams were obtained two hours following the 99mTc-PYP injection in four patients. Sequential Polaroid scintigrams were obtained with a Pho/Gamma Camera H.P. (Searle Radiographics) utilizing a 15,000 hole

References
low energy all purpose collimator (LEAP-parallel). There was no appreciable difference in the quality of the resolution of Polaroid scintigrams obtained by the LEAP collimator and scintigrams obtained with a high resolution collimator (H.R. parallel) in three patients studied with both collimators.

Scintigrams were classified as diffuse when the uptake was generalized over the entire left ventricle and localized when the uptake was limited to one discrete region of the left ventricle. Intensity was graded from 0 to 5+ according to the scheme devised by Parkey et al. Zero and 1+ were considered negative. The site of localized uptake was recorded as anterior, inferior, lateral or posterior. All scintigrams were interpreted by two independent observers with no prior knowledge of the clinical status of the patient. Since all patients included in this study are still alive, postmortem histologic data are not available.

Twenty-eight patients with acute myocardial infarction (mean age 53 ± (SE) 7.2 yr) were studied with serial electrocardiograms and serum enzymes (CPK, SGOT, LDH) over the first three days of their hospitalization. Scintigrams were performed two to three days after the onset of symptoms.

Patients without clinical evidence of acute myocardial infarction manifested no acute changes in electrocardiograms obtained immediately prior to the scintigraphic study, and serum CPK, SGOT, and LDH enzymes, measured in patients with angina pectoris, were within normal limits. Based on the left ventricular wall motion pattern as seen on left ventricular cineangiogram, these patients were divided into three groups. Group I consisted of ten patients (mean age 51.2 ± 2.6 yr) with areas of localized left ventricular dysfunction. Group II contained five patients (mean age of 53.0 ± 3.9 yr) with areas of left ventricular akinesis and hypokinesis, and Group III, seven patients (mean age of 51.4 ± 3.0 years) with normal left ventricular wall motion. Left ventricular volume in these patients was estimated from single plane right anterior oblique 35 mm cineangiograms using the method of Kasser and Kennedy.

Results

In the 28 patients with characteristic electrocardiographic and enzyme evidence of acute myocardial infarction, myocardial scintigrams displayed localized activity in 15 patients with transmural, and diffuse activity in 13 patients with subendocardial myocardial infarction. The area of scintigraphic activity in patients with acute transmural myocardial infarction corresponded closely to the location of myocardial infarction by electrocardiogram. A typical scintigram in a patient with acute anterolateral transmural myocardial infarction is shown in figure 1.

In Group I (summarized in table 1), five patients had evidence of mild left ventricular failure and three patients had stable angina pectoris at the time of the study. Each patient had persistent ST-segment elevation characteristic of left ventricular aneurysm. The angiographic left ventricular volume was abnormally large in all but one patient. The mean end-diastolic and end-systolic volumes were 112.3 ± 14.9 ml/m² and 77.3 ± 16 ml/m², respectively. The four patients in this group who were studied after cardiovascular

### Table 1. Patients with Left Ventricular Dysfunction (Group I)

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age (yr)</th>
<th>Sex</th>
<th>Myocardial scintigraphy</th>
<th>Coronary artery graft surgery</th>
<th>Coronary angiography</th>
<th>Angina pectoris</th>
<th>Cardiomyopathy</th>
<th>LV angiographic localization of infarction</th>
</tr>
</thead>
<tbody>
<tr>
<td>D.K.</td>
<td>45/M</td>
<td></td>
<td>3+ Anterolateral</td>
<td></td>
<td></td>
<td>No</td>
<td>No</td>
<td>LV anterior</td>
</tr>
<tr>
<td>B.S.</td>
<td>46/M</td>
<td></td>
<td>3+ Anterolateral</td>
<td></td>
<td></td>
<td>No</td>
<td>No</td>
<td>LV anterior</td>
</tr>
<tr>
<td>E.Z.</td>
<td>58/M</td>
<td></td>
<td>3+ Anterolateral</td>
<td></td>
<td></td>
<td>No</td>
<td>No</td>
<td>LV anterior</td>
</tr>
<tr>
<td>W.K.</td>
<td>60/F</td>
<td></td>
<td>3+ Anterolateral</td>
<td></td>
<td></td>
<td>No</td>
<td>No</td>
<td>LV anterior</td>
</tr>
<tr>
<td>L.N.</td>
<td>49/M</td>
<td></td>
<td>3+ Anterolateral</td>
<td></td>
<td></td>
<td>No</td>
<td>No</td>
<td>LV anterior</td>
</tr>
<tr>
<td>F.P.</td>
<td>50/M</td>
<td></td>
<td>3+ Anterolateral</td>
<td></td>
<td></td>
<td>No</td>
<td>No</td>
<td>LV anterior</td>
</tr>
<tr>
<td>J.C.</td>
<td>35/F</td>
<td></td>
<td>3+ Anterolateral</td>
<td></td>
<td></td>
<td>No</td>
<td>No</td>
<td>LV anterior</td>
</tr>
<tr>
<td>H.T.</td>
<td>50/M</td>
<td></td>
<td>3+ Anterolateral</td>
<td></td>
<td></td>
<td>No</td>
<td>No</td>
<td>LV anterior</td>
</tr>
<tr>
<td>J.L.</td>
<td>40/M</td>
<td></td>
<td>3+ Anterolateral</td>
<td></td>
<td></td>
<td>No</td>
<td>No</td>
<td>LV anterior</td>
</tr>
</tbody>
</table>

* LV angiogram obtained before and after surgery.
surgery had persistent localized left ventricular dyskinesis by left ventriculography. Roentgenographic and fluoroscopic examination revealed calcification of the left ventricular wall in four patients. The mean duration from the day of myocardial infarction to the time of imaging for the entire group was 32.1 ± 9.8 months and in four patients studied after cardiovascular surgery, 28.5 ± 9.0 months from the day of the operation. One patient had diffuse uptake. The remaining nine patients in this group had intense focal uptake, localized in each case to the site of left ventricular dyskinesis. Follow-up scintigrams obtained in five patients, two to eight weeks later, gave identical results. Typical serial scintigrams obtained eight weeks apart and left ventricular cineangiogram in a patient with apical left ventricular dyskinesis are shown in figures 2 and 3, respectively. Repeat scintigrams obtained one hour following the initial imaging and approximately two hours following the $^{99m}$Tc-PYP injection showed persistence of activity in the area of uptake along with increased uptake in the bones. Figure 4 shows scintigrams in a patient with left ventricular aneurysm obtained one and two hours following $^{99m}$Tc-PYP injection.

Group II is summarized in table 2. All patients in this group had localized hypokinesis or akinesis. None had electrocardiographic findings suggestive of ventricular aneurysm. Left ventricular end-diastolic and end-systolic volumes were $77.0 \pm 9.6$ ml/m$^2$ and $35.16 \pm 7.6$ ml/m$^2$, respectively. Three patients had a history of previous myocardial infarction. Two had positive scintigrams. The mean time of imaging following the myocardial infarction in these two patients was 9.5 months. None of the patients in this group had roentgenographically visible calcification in the left ventricular wall. All patients had stable angina pectoris.
toris. In the two patients with positive scintigrams, the frequency of anginal episodes averaged three times per week in one patient and four to five times a month in the other, readily relieved by rest and nitroglycerin in both.

Table 3 summarizes the seven patients in Group III who had normal left ventricular wall motion, normal left ventricular volumes and negative myocardial scintigrams. None of the patients in this group had previous myocardial infarction. Left ventricular end-diastolic and end-systolic volumes were 75.3 ± 2.3 ml/m² and 21.0 ± 3.0 ml/m², respectively. Left ventricular wall calcification was not detected in any of these patients.

Discussion

The bone scanning agent technetium 99m stannous pyrophosphate has recently been applied to the diagnosis of acute myocardial infarction. Characteristic patterns of localized uptake in acute transmural, and diffuse uptake in acute subendocardial myocardial infarction have been described. Mild diffuse uptake has been reported in dilated hearts secondary to cardiomyopathy and in a few patients with chest pain but without the usual clinical evidence of acute myocardial infarction. Localized intense uptake at the site of infarction has been reported to be characteristic of acute transmural myocardial infarction and some cases of subendocardial infarction.

Our observation that all 28 patients with acute myocardial infarction had positive scintigrams, with localized activity in the 15 patients with acute transmural and diffuse activity in the 13 patients with acute subendocardial myocardial infarction, is in agreement with previous reports. However, in addition, 11 of 15 patients with previous myocardial infarction but without clinical, electrocardiographic or enzymatic evidence of acute myocardial infarction had positive scintigrams. Uptake was localized to the angiographically visualized site of left ventricular wall motion abnormality in each case. The presence of elevated left ventricular end-diastolic volume in 10 of 11 patients with localized positive scintigrams suggests that an appreciably large area of left ventricle must be involved in order for the scintigram to develop this pattern. Patients without acute myocardial infarction who had normal left ventricular wall motion had negative scintigrams, irrespective of the presence or absence of a previous myocardial infarction.

Our data do not offer a ready explanation for the mechanism of uptake of 99mTc-PYP by these presumably...
relatively avascular and fibrotic areas of the left ventricle. Calcium ions are known to localize within the mitochondria of myocardial cells in steroid-induced focal necrosis, incorporating into a crystalline structure thought to be hydroxyapatite.9,10 Calcium has also been demonstrated histologically in the irreversibly damaged myocardium in the infarcted area.10 The uptake of 99mTc-PYP by these mineral deposits in the infarcted area is considered to be the likely process leading to the development of the positive scintigrams.2 A period of reflow following the irreversible cellular injury has been considered essential for calcium uptake during experimental myocardial infarction.11 Buja and his colleagues15 have recently reported maximal concentration of the 99mTc-PYP along the margins of the experimentally-induced infarcted area corresponding to the zone of maximal calcium deposition. These authors conclude that 99mTc-PYP localization in the infarcted area is a function of the collateral blood flow in the various regions of acute myocardial infarction.

Four of our patients with positive scintigrams without acute myocardial infarction had radiographically visible calcification in the area of the uptake, and a higher incidence could possibly be demonstrated by histologic techniques.13 Deposition of calcium in the left ventricular myocardium may occur in patients following cardiovascular surgery.14,15 These observations suggest the possible role of uptake of 99mTc-PYP by dystrophic calcification in the area of previous myocardial necrosis. In all four of our patients studied after cardiovascular surgery, imaging was performed several months after the surgical intervention, thus precluding acute myocardial infarction in the intraoperative or immediate postoperative phase as the cause of the positive scintigrams.

Persistence of the same degree of scintigraphic activity in five patients two to eight weeks following the initially positive scintigram makes the possibility of a clinically silent acute myocardial infarction an unlikely event to explain the 99mTc-PYP uptake, since uptake usually disappears or becomes less intense sooner than this following an acute infarction in the majority of patients.1,2 It is unlikely that scintillation of an intracardiac blood pool could explain our findings, since scintigrams repeated one hour following the initial imaging and two hours following the 99mTc-PYP injection in patients with left ventricular aneurysm remained positive. In addition, scintigrams of our Group III patients with normal left ventricular wall motion were uniformly negative.

Willerson et al.5 have reported faintly positive scintigrams in patients with unstable angina pectoris and in some patients with other types of chest pain without evidence of acute myocardial infarction, thus raising the possibility that 99mTc-PYP myocardial scintigrams may be able to detect severe myocardial ischemia in the absence of infarction. Only thirty percent of our patients with left ventricular aneurysm with positive scintigrams had stable angina pectoris and the remaining were angina free. Our two patients with left ventricular akinesia and positive scintigrams also had stable angina, making the presence of myocardial necrosis or severe ischemia unlikely as the basis for the 99mTc-PYP uptake.

Persistent ST-segment elevation in the electrocardiogram of patients with left ventricular aneurysm, present in

Table 2. Patients with Left Ventricular Hypokinesis and/or Akinesis (Group II)

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age/sex (yr)</th>
<th>Angina pectoris</th>
<th>Q wave</th>
<th>ST depression</th>
<th>LV angiographic localization of hypokinesis</th>
<th>Akinetis</th>
<th>Time myocardial scintigram performed after infarction (months)</th>
<th>Myocardial scintigram</th>
</tr>
</thead>
<tbody>
<tr>
<td>T.R.</td>
<td>56/M</td>
<td>+</td>
<td>V1-V5</td>
<td>II, III, AVF</td>
<td>Anterior</td>
<td>Inferior</td>
<td>120</td>
<td>N.U.</td>
</tr>
<tr>
<td>A.H.</td>
<td>44/M</td>
<td>+</td>
<td>0</td>
<td>I, II, AVL</td>
<td>V3-V6</td>
<td>Apical</td>
<td>0</td>
<td>N.I.</td>
</tr>
<tr>
<td>E.B.</td>
<td>58/M</td>
<td>+</td>
<td>0</td>
<td></td>
<td>Anterolateral</td>
<td>0</td>
<td>N.I.</td>
<td>N.U.</td>
</tr>
<tr>
<td>D.V.</td>
<td>52/M</td>
<td>+</td>
<td>V1</td>
<td>V2-V6</td>
<td>Anterior</td>
<td>Apical</td>
<td>12</td>
<td>2+ Anterolateral</td>
</tr>
<tr>
<td>R.F.</td>
<td>54/M</td>
<td>+</td>
<td>V2-V6</td>
<td></td>
<td>Anterolateral</td>
<td>0</td>
<td>7</td>
<td>3+ Anterolateral</td>
</tr>
</tbody>
</table>

N.I. = No evidence of previous myocardial infarction.
N.U. = No significant uptake.

Table 3. Patients with Normal Left Ventricular Wall Motion (Group III)

<table>
<thead>
<tr>
<th>Patient</th>
<th>Age/sex (yr)</th>
<th>Angina pectoris</th>
<th>Q wave</th>
<th>ST depression</th>
<th>Time myocardial scintigram performed after infarction (months)</th>
<th>Myocardial scintigram</th>
</tr>
</thead>
<tbody>
<tr>
<td>J.R.</td>
<td>62/M</td>
<td>+</td>
<td>I, AVL</td>
<td>AVL</td>
<td>96</td>
<td>N.U.</td>
</tr>
<tr>
<td>R.C.</td>
<td>59/M</td>
<td>+</td>
<td>V1-V2</td>
<td></td>
<td>V6-V9</td>
<td>N.I.</td>
</tr>
<tr>
<td>D.B.</td>
<td>40/M</td>
<td>+</td>
<td>0</td>
<td>V1-V6</td>
<td>N.I.</td>
<td>N.I.</td>
</tr>
<tr>
<td>W.B.</td>
<td>33/M</td>
<td>+</td>
<td>II, III, AVF</td>
<td>1, II, III</td>
<td>12</td>
<td>N.I.</td>
</tr>
<tr>
<td>T.W.</td>
<td>40/M</td>
<td>+</td>
<td>III</td>
<td>0</td>
<td>24</td>
<td>N.U.</td>
</tr>
<tr>
<td>R.S.</td>
<td>41/M</td>
<td>+</td>
<td>0</td>
<td>I, AVL</td>
<td>V5-V6</td>
<td>N.I.</td>
</tr>
<tr>
<td>G.K.</td>
<td>62/M</td>
<td>+</td>
<td>V1-V6</td>
<td>0</td>
<td>12</td>
<td>N.U.</td>
</tr>
</tbody>
</table>

N.I. = No evidence of previous myocardial infarction.
N.U. = No significant uptake.
all of our patients with left ventricular dyskinesis, has been ascribed by some authors to persistent ischemia in the region of the left ventricular aneurysm. The possible role of this presumed ischemic zone in the \textsuperscript{99m}Tc-PYP uptake in our patients cannot be determined. However, two patients in Group II with \textsuperscript{99m}Tc-PYP uptake in akinetic areas did not manifest ST-segment elevation.

Our findings indicate that if there is a history of a previous myocardial infarction with left ventricular aneurysm, especially in patients with large areas of left ventricular dyskinesis, a positive localized \textsuperscript{99m}Tc-PYP myocardial scintigram should not be interpreted as conclusive evidence of acute myocardial infarction.

Acknowledgment

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References

Technetium 99m stannous pyrophosphate myocardial imaging in patients with and without left ventricular aneurysm.
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