Localization of $^{99m}$Tc-Sn-Pyrophosphate in Left Ventricular Aneurysms


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Concentration of $^{99m}$Tc-pyrophosphate ($^{99m}$Tc-PP) in the area of left ventricular aneurysms is discussed. In three cases clinical, laboratory, and electrocardiographic (ECG) evidence did not indicate that these patients currently had an acute myocardial infarction, but each patient had a clinical history and an ECG picture compatible with an old myocardial infarction. Cardiac catheterization revealed a large left ventricular aneurysm in all three cases. The reason for the preferential uptake of the radionuclide in the area of these aneurysms is not certain at this time.


Since the successful production of scintillation camera images of recent myocardial infarctions using $^{99m}$Tc-Sn-pyrophosphate ($^{99m}$Tc-PP) by Bonte et al. (1), several clinical investigators have studied the usefulness of this procedure (2-5). These investigators have shown a remarkable affinity of $^{99m}$Tc-labeled phosphate compounds for both acute transmural (1-4) and acute subendocardial (5) myocardial infarctions. Technetium-$^{99m}$pyrophosphate appears to be one of the most effective of the $^{99m}$Tc-labeled phosphate compounds for this procedure (6). The number of false-positive and false-negative studies reported so far is impressively small (3,5,7). False positives may be caused by old rib fractures (5), metastatic carcinoma (7), breast tumors (8), myocardial contusion (9), myocardiopathies (10), severe ischemia (10), and ventricular aneurysms (11). False-negative scans may result from delay of the scintigrams until after the first week after infarction (12,13).

During a recent clinical trial of pyrophosphate myocardial scanning at our institution (14) and following a discussion with Dr. August Miale (Miami, Florida), it came to our attention that discretely positive scintigrams were obtained in some patients with left ventricular aneurysms. This report discusses three patients with this phenomenon.

CASE REPORTS

Case 1. A 48-year-old white man was admitted after experiencing the onset of “chest tightness,” with nausea but without vomiting, shortness of breath, or diaphoresis. The pain was similar to that experienced about 2.5 years before admission, during an uncomplicated acute myocardial infarction. The patient had done well since that episode, and his history and physical examination were otherwise unremarkable. He was admitted with a presumptive diagnosis of acute myocardial infarction.

Chest x-ray and serum enzymes (including creatine phosphokinase, CPK MB isoenzyme) were normal during the patient's hospitalization. Serial ECGs indicated an old anteroseptal myocardial infarction and anterolateral ischemia, but no evolutionary changes.

A $^{99m}$Tc-PP scan was performed 1 day after admission in order to rule out an acute myocardial infarction. This scintigram (Fig. 1) revealed an area of localized uptake of radionuclide in the anterior portion of the left ventricle, consistent with an acute myocardial infarction. Cardiac catheterization was performed at the end of 1 week and revealed a left ventricular aneurysm involving the anterior wall and apex of the heart, and total occlusion of the left anterior descending coronary artery. No mural thrombus was identified. A $^{99m}$Tc-PP, myocardial scan was repeated 1 day after the cardiac catheter-
Left ventricular wall motion was evaluated by analysis of the first-transit left ventricular radionuclide data (Fig. 2). This revealed a dyskinetic anterior ventricular wall consistent with an aneurysm. Discrete uptake of the radionuclide was also noted in this region on the static images obtained 1 hr after injection. The positive area on static images was thought to correspond with that of the left ventricular aneurysm.

**Case 2.** A 61-year-old white man was admitted on Feb. 12, 1976, because of unresponsive cardiac arrhythmias and the suspicion of a left ventricular aneurysm. He had suffered an acute posterior myocardial infarction in 1965 and had reinfarcted anterolaterally in November 1975. He denied chest pain since that time but admitted experiencing diminishing exercise tolerance and increasing shortness of breath. The patient's remaining history and physical examination were unremarkable.

Chest x-ray and serum enzymes (including CPK MB isoenzyme) failed to confirm a recent myocardial infarction. Serial ECGs were stable and revealed an old extensive anterior myocardial infarction with persistent ST-segment elevation suggestive of a left ventricular aneurysm. Multifocal PVCs and left atrial enlargement were also indicated by the ECG.

Five days after admission, the patient underwent cardiac catheterization, which revealed a large anterior left ventricular aneurysm and three-vessel coronary artery disease. Two days later 99mTc-PP1 myocardial scan (Fig. 3) revealed a large area of increased activity in the anterolateral segment of the left ventricle, corresponding to the location of the ventricular aneurysm.

The patient subsequently underwent open-heart surgery for aortocoronary saphenous-vein by-pass grafting and a left ventricular aneurysmectomy. Microscopic examination of the surgical specimen showed the aneurysm to be composed largely of scar tissue containing foci of hemosiderin and nests of myocardial cells. A portion of mural thrombus was also included. A repeat 99mTc-PP1 scan 24 days after surgery failed to show the previously noted uptake of radionuclide and was considered normal (Fig. 4).

**Case 3.** A 54-year-old white man with cardiac arrhythmias that were unresponsive to antiarrhythmic drug management was transferred to our institution. He complained of parasternal chest tightness with radiation to his left shoulder and arm, but denied syncopal episodes. The patient had suffered an acute myocardial infarction 15 years before admission and had had acute rheumatic fever as a child. History and physical examination were otherwise unremarkable. He was admitted for diagnosis and treatment of the arrhythmias and to rule out an acute myocardial infarction. The chest x-ray showed mild cardiomegaly with apical cardiac calcification, and mild pulmonary–venous hypertension. Serum enzymes (including CPK MB isoenzyme) were within normal limits. The ECG revealed paroxysmal ventricular tachycardia with arteriovenous dissociation, evidence of an old anterior myocardial infarction, inferolateral myocardial ischemia, and suggestion of a left ventricular aneurysm. No evolutionary changes were present on serial ECGs.

Cardiac catheterization performed eight days after admission revealed a large anterolateral left ventricular aneurysm with calcification of the wall, and an apical mural thrombus. A totally occluded left anterior descending coronary artery was also seen. A
myocardial study (Fig. 5) was performed 13 days after admission. Paradoxical motion of the left ventricular wall was shown by first-transit data analysis. Increased accumulation of the radionuclide in the left ventricular myocardium was also seen on the static images taken 1 hr later. The patient subsequently underwent aneurysmectomy. Microscopically, the aneurysm consisted of fibrous scar tissue with small nests of myocardium.

**DISCUSSION**

The introduction of $^{99m}{\text{Tc}}$-PP$_1$ as an agent to image acute myocardial infarction has added a new dimension to myocardial scanning, and many reports have appeared describing the sensitivity and specificity of this agent for recent myocardial infarcts (1-6). The paucity of reported false-positive and false-negative scintigrams lends additional support to the value of this test in the diagnosis of acute myocardial infarction. During our recent clinical evaluation of $^{99m}{\text{Tc}}$-PP$_1$, for myocardial imaging (14), we noted several false-positive examinations, and after consultation with Dr. August Miale, whose group had noted the accumulation of $^{99m}{\text{Tc}}$-PP$_1$ in ventricular aneurysms, we decided to investigate these cases.

All three patients had a history of a prior myocardial infarction as well as ECG evidence suggestive of a left ventricular aneurysm. In all cases, ECG and serum enzyme data indicated that none had suffered from an acute myocardial infarction to account for current symptoms. In each patient, a $^{99m}{\text{Tc}}$-PP$_1$ myocardial scan was discretely positive, and in two cases motion studies showed dyskinesia of the ventricular wall. In addition, every patient gave confirmation by cardiac catheterization of a left-ventricular aneurysm. Although the scintigrams followed catheterization by 2-5 days in two of our patients, we do not feel that the uptake of $^{99m}{\text{Tc}}$-PP$_1$ was related to any possible subclinical myocardial trauma secondary to the catheterization procedure. Patients without ventricular aneurysms who had uncomplicated cardiac catheterization prior to $^{99m}{\text{Tc}}$-PP$_1$ studies showed no abnormal ventricular activity.

The reason for the discrete localization of $^{99m}{\text{Tc}}$-PP$_1$ in the area of these ventricular aneurysms is uncertain at this time, but it may be related to the influx of calcium into ischemic myocardial tissue, reported earlier by Shen and Jennings (15). Since the aneurysmal ventricular wall consists mainly of fibrous scar tissue interspersed with minute areas of ischemic myocardium, calcium accumulation might result following arterial reflow in the ischemic regions. This mechanism is supported by the occasional occurrence of extensive calcification of the myocardial scar in old infarcts and ventricular aneurysms. Thus, patients with left ventricular aneurysms characterized by persistent chronic ischemia might experience calcium influx into the ischemic regions of myocardium, and they might thereby exhibit consistently positive $^{99m}{\text{Tc}}$-PP$_1$ studies on serial examinations, as noted in two of the cases. The constant pattern of radio- nuclide uptake differs markedly from the gradual decrease in activity seen in serial scans of patients with acute myocardial infarctions (1,4,14). This possible mechanism is compatible with temporal and topographic events depicted by Buja et al. (16) with regard to calcium influx and pyrophosphate uptake. Alternatively, calcium accumulation or nonspecific protein binding along the interface between a mural thrombus and the ventricular wall might result in the accumulation of the radionuclide. However, without definitive localization of $^{99m}{\text{Tc}}$-PP$_1$, in pathologic specimens, these theories remain speculative.

Localized uptake of $^{99m}$Tc-labeled phosphate compounds seems to occur in conditions other than re-
cent acute myocardial infarctions: breast tumors (8), myocardial contusion (9), rib fractures, myocardopathies (10), and severe unstable atherosclerotic heart disease without infarction (10). Moreover, recent evidence indicates that ventricular aneurysms (11), metastatic carcinoma (17), penetrating myocardial trauma (17), and countershock used during cardiopulmonary resuscitation (18) might result in false-positive studies. Our observations have been presented to confirm ventricular aneurysms as another potential cause of false-positive \(^{113}\text{Tc}\)-PP, myocardial scans. Caution is urged in the interpretation of such positive scintigrams when evidence of left ventricular aneurysm is present.

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REFERENCES


