Intense Myocardial Uptake of ^{99m}Tc-Diphosphonate in a Uremic Patient with Secondary Hyperparathyroidism and Pericarditis: Case Report

Warren R. Janowitz and Aldo N. Serafini

Baumritter Institute of Nuclear Medicine, Mount Sinai Medical Center, Miami Beach, Florida

A patient on chronic hemodialysis, with secondary hyperparathyroidism and uremic pericarditis, showed marked myocardial uptake of ^{99m}Tc-diphosphonate. Several possible mechanisms for the myocardial uptake are discussed.

J Nucl Med 17: 896–898, 1976

Myocardial scintigraphy with ^{99m}Tc-labeled bone-imaging agents, primarily ^{99m}Tc-pyrophosphate, is utilized increasingly for detecting and sizing acute myocardial infarcts (*1*). Other causes of myocardial uptake of these agents, including myocardial contusion and dc countershock cardioversion, have also been described. Uptake in the myocardial region in two patients with carcinoma has been reported (*2*), but without any definite cause being established.

Primary and secondary hyperparathyroidism have also been shown to alter the skeletal distribution of bone-seeking agents (*3*). Soft-tissue uptake has appeared in bone scans in patients with hyperparathyrodism (*4*), myositis ossificans (*5*), metastatic calcification (*6*), cerebral infarction, benign and malignant breast lesions, bronchogenic carcinoma, and other conditions. The following case report describes the intense myocardial uptake found in a patient on chronic dialysis, with secondary hyperparathyroidism and uremic pericarditis; there was no evidence of acute myocardial infarction.

CASE REPORT

A 42-year-old man was admitted on Nov. 19, 1975, with the chief complaints of chest pain, nausea, vomiting, and jaundice. In March 1974, after an episode of pulmonary edema, he had been found to be in renal failure, thought to be secondary to uncontrolled hypertension. In September 1974, chronic hemodialysis was started. At that time his blood urea nitrogen level was 138 mg% (normal 10–20), creatinine 12.7 mg% (normal 0.7–1.5), calcium 7.7 mg% (normal 8.5–10.5), and inorganic phosphate of 7.0 mg% (normal 2.5–4.5). In September 1975, he was readmitted with jaundice and HAA+ hepatitis. At that time he was found to be hypercalcemic, with a calcium level 12.2–12.5 mg% and an inorganic phosphate level of 7.6 mg%. The serum parathyroid hormone level was up to 955 μl eq/ml (normal 10–60). There was also evidence of dense calcification in the radial arteries.

At the present admission, the patient was still clinically jaundiced and was found to have a pericardial friction rub, with serial electrocardiograms suggestive of pericarditis. The serum CPK was never above normal limits. The SGOT and LDH had been elevated previously secondary to his hepatitis. The patient's serum calcium level ranged over 12.2–13.6 mg%, with inorganic phosphate 5.4–7.3 mg%.

Three weeks after admission, the patient's clinical status had improved, with disappearance of the chest pain and friction rub, and a bone scan was obtained for evaluation of the hypercalcemia. An anterior chest scintigraph, obtained 4 hr after injection of 15 mCi of ^{99m}Tc-diphosphonate, showed marked uptake of the radiopharmaceutical within the myocardium and diffuse increased uptake in the lungs (Fig. 1). A posterior view also showed a localized area of uptake within the upper right chest. No other bony abnormalities or soft-tissue uptake was seen. No abnormal calcifications were found on chest x-ray,

Received April 8, 1976; revision accepted May 7, 1976.

For reprints contact: Warren R. Janowitz, Nuclear Medicine Branch, National Naval Medical Center, Bethesda, MD 20014.

and a skeletal survey showed no bone changes characteristic of hyperparathyroidism. Additional calcification of the peripheral blood vessels was seen.

Because of the findings of the first bone scan, a myocardial study with 2 mCi of ^{201}Tl was obtained 9 days later. This was immediately followed by a repeat study with ^{99m}Tc-diphosphonate, with the images obtained 90 min after injection. Figure 2 shows the anterior and 45° LAO ^{201}Tl and ^{99m}Tc-diphosphonate scintigrams. The distribution of the two agents is almost identical, confirming that the ^{99m}Tc-diphosphonate activity was within the myocardium.

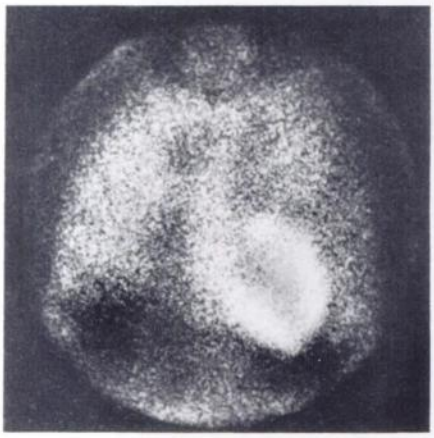

FIG. 1. Anterior chest scintigram taken 4 hr after injection of ^{99m}Tc-diphosphonate. Note marked uptake in heart with hazy diffuse uptake in both lungs.

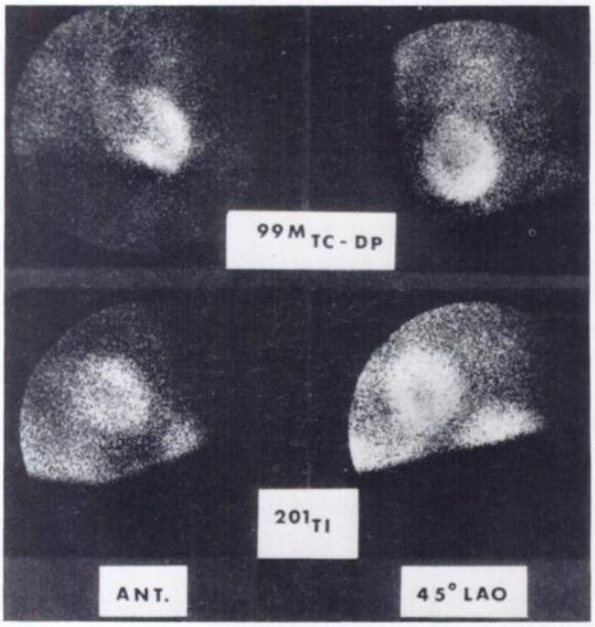

FIG. 2. (Top) Anterior and 45° LAO ^{99m}Tc-diphosphonate scintigrams obtained 9 days after that shown in Fig. 1. (Bottom) Corresponding views obtained on same day with ^{201}Tl.

The patient was discharged several days later and no followup scans have been obtained.

DISCUSSION

Chemoabsorption of the ^{99m}Tc-Sn-phosphate complex onto the calcium of hydroxyapatite is the widely accepted view of the uptake mechanism of these agents in skeletal imaging. In the acutely infarcted myocardium, hydroxyapatite formation in the mitochrondria has been postulated as the mechanism of diphosphonate uptake (*7*).

Conger et al. (*8*) reporting on a series of patients on chronic dialysis, found a 60% incidence of pulmonary calcification. However, such visceral calcifications (lung, heart, or skeletal muscle) did not show ^{99m}Tc-diphosphonate uptake in patients with chronic uremia and metastatic calcification (*4*). This finding has been attributed to the relatively recent observation that there are two types of pathologic calcification in patients with uremia: (A) hydroxyapatite formation in tumoral or periarticular calcifications; and (B) deposits in the visceral organs of an amorphous or microcrystalline compound composed of calcium, magnesium, and phosphorus with a diffraction pattern and molar ratio very similar to those of magnesium whitlockite (*9*). These two forms of calcium deposits are markedly different in their in vitro binding of ^{99m}Tc-diphosphonate (*4*). Previously, most authors assumed that all tissue calcification in uremic patients was hydroxyapatite.

In our patient, the presence of uremic pericarditis is a complicating factor in evaluating the myocardial uptake. There are several reasons, however, why pericarditis or accompanying myocarditis is less likely to be the primary cause. The pattern of ^{99m}Tc-diphosphonate uptake seen in Fig. 2 is almost identical to that of ^{201}Tl, suggesting that the diphosphonate is distributed throughout the myocardium. Uremic pericarditis may be associated with myocarditis, which usually is located adjacent to the inflamed pericardium (*10*). Myocarditis is also associated with hypotension, shock, arrythmias, and heart failure, and these findings were not a prominent feature of our patient's illness. The fact that his scans were obtained 3 and 4 weeks after admission, at a time when his pericarditis and clinical status were improving, also makes myocarditis less likely as a causative factor.

No clinical evidence was found to support the hypothesis that a myocardial infarction was present. Moreover, the thallium images showed no transmural defects. While subendocardial infarcts may present with diffuse ^{99m}Tc-pyrophosphate uptake, they typically show much less activity. Nor is there any reason to implicate the patient's hepatitis-B anti-

genemia as a contributing cause. Thus, secondary hyperparathyroidism seems to be the most likely cause. The lung and posterior thoracic uptakes also point to a more widespread process than could be attributed to pericarditis–myocarditis.

In view of the previously cited work of Conger et al. (*8*), it would be of great interest to know the nature of the metastatic calcifications in this patient. Although this is only speculation, this patient may have hydroxyapatite in his visceral calcifications instead of the supposedly more common amorphous deposits. This would explain why myocardial ^{99m}Tc-phosphate uptake has not been previously described in uremic patients. We do not know why this would occur in our patient.

REFERENCES

1. Bonte FJ, Parkey RW, Graham KD, et al.: A new method for radionuclide imaging of myocardial infarcts. *Radiology* 110: 473–474, 1974

2. Soin JS, Burdine JA, Beal W: Myocardial localization of ^{99m}Tc-pyrophosphate without evidence of acute myocardial infarction. *J Nucl Med* 16: 944–946, 1975

3. Sy WM: Bone scan in primary hyperparathyroidism. *J Nucl Med* 15: 1089–1091, 1974

4. Conger JD, Alfrey AC: Letter to editor. *Ann Intern Med* 84: 224–225, 1976

5. Suzuki Y, Hisada K, Masanori T: Demonstration of myositis ossificans by ^{99m}Tc-pyrophosphate bone scanning. *Radiology* 111: 663–664, 1974

6. Richards AG: Metastatic calcification detected through scanning with ^{99m}Tc-polyphosphate. *J Nucl Med* 15: 1057–1060, 1974

7. Buja LM, Parkey RW, Dees JH, et al.: Morphologic correlates of Tc-99m stannous pyrophosphate imaging of acute myocardial infarcts in dogs. *Circulation* 52: 596–607, 1975

8. Conger JD, Hammond WS, Alfrey AC, et al.: Pulmonary calcification in chronic dialysis patients: Clinical and pathologic studies. *Ann Intern Med* 83: 330–336, 1975

9. Contiguglia SR, Alfrey AL, Miller NL, et al.: Nature of soft tissue calcification in uremia. *Kidney Int* 4: 229–235, 1973

10. Compty CM, Cohen SL, Shapiro FL: Pericarditis in chronic uremia and its sequels. *Ann Intern Med* 75: 174–183, 1971