Technetium-99m-Sn-Pyrophosphate Pharmacokinetics and Bone Image Changes in Parathyroid Disease

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Skeletal abnormalities in 12 patients with primary hyperparathyroidism, five patients with pseudohypoparathyroidism, and three patients with hypoparathyroidism were studied to compare the diagnostic sensitivity of bone radiologic examination to that of radionuclide studies using \(^{99m}\text{Tc-Sn-pyrophosphate} (^{99m}\text{Tc-PP})\), a skeletal-seeking radiopharmaceutical. The results were compared with bone mineral content as measured by the Norland-Cameron densitometer. Kinetic data of the blood disappearance and plasma clearance of \(^{99m}\text{Tc-PP}\), were obtained and compared with data of control subjects without evidence of parathyroid disease. Bone imaging with \(^{99m}\text{Tc-PP}\) may be more sensitive than routine skeletal radiographs and bone mineral analysis for the evaluation of skeletal abnormalities in patients with parathyroid dysfunction. The enhanced plasma clearance of the tracer observed in patients with primary hyperparathyroidism may reflect the direct effect of excessive parathyroid hormone on the renal handling of \(^{99m}\text{Tc-Sn-pyrophosphate}\).


Bone imaging with \(^{99m}\text{Tc}\)-labeled phosphate compounds has been employed most extensively for the detection of metastatic bone lesions, primary bone tumors, infections, Paget's disease, and other benign lesions of bone (1–5). Application of these imaging techniques for the evaluation, diagnosis, and study of metabolic bone disease has been limited. The present studies were undertaken to establish the role of bone imaging in determining bone abnormalities in patients with primary hyperparathyroidism, hypoparathyroidism, and pseudohypoparathyroidism. In a large series of patients, skeletal radiographs indicated that focal skeletal lesions (e.g., Brown tumors, cysts, cortical or subperiosteal resorption) occur in approximately 10% of patients with primary hyperparathyroidism whereas less specific abnormalities such as generalized demineralization may be found in 30% (6). A comparison of the relative sensitivity of bone roentgenography to that of bone imaging in the study of metabolic bone disease may provide additional information.

In the present studies scintillation imaging was performed with \(^{99m}\text{Tc-Sn-pyrophosphate} (^{99m}\text{Tc-PP})\). Urinary excretion of unlabeled pyrophosphate has been reported to be increased in patients with primary hyperparathyroidism (7). Contradictory results, however, have been reported by others (8). Thus, knowledge concerning the blood disappearance and renal clearance of \(^{99m}\text{Tc-PP}\) may be of diagnostic value.

MATERIALS AND METHODS

The specific parathyroid disorder in each patient was established by clinical, biochemical, and radiologic evaluation, including RIA determination of serum parathormone levels. Twelve patients with
primary hyperparathyroidism were studied. Seven of these were confirmed by histologic examination of the lesion and the remaining five patients by generally accepted criteria for the diagnosis of primary hyperparathyroidism (including hypercalcemia, elevated parathyroid hormone levels, abnormal responses to oral orthophosphate loading, and histologic evidence of hyperparathyroidism from iliac crest bone biopsy). Three patients had developed hypoparathyroidism following surgery for either thyroid or parathyroid disease (Table 1). The duration of hypoparathyroidism in these patients ranged from 7 to 11 years. Each was maintained in a normocalcemic state with regular doses of 100,000–200,000 units of vitamin D$_2$ and oral calcium supplements, but all had been off treatment for 1–3 months at the time of bone imaging and kinetics studies. In five patients with pseudohypoparathyroidism (Table 1), the diagnosis had been confirmed by clinical features (hypocalcemia, elevated serum PTH levels, brachydactyly) or the failure to enhance urinary excretion of 3',5'-cyclic adenosine monophosphate during an infusion of parathyroid extract. None of these patients was under vitamin D therapy at the time of these studies.

Bone mineral content was evaluated by means of the Norland-Cameron bone mineral analyzer. Iodine-125 (400 mCi) was used as the external radiation source. Radiation, collimated to 3 mm, was passed through the radius of the nondominant arm and detected on the other side of the radius with the scintillation detector. The selected arm was repositioned, studied two or three times, and a mean value was calculated. Bone mineral content (gm/cm) and bone width (cm) were measured and their ratio (gm/cm$^2$) used for analysis. These results were compared with the normal values for age- and sex-matched American Caucasians and considered abnormal if a measurement fell below the 95% confidence limit (9).

Bone roentgenograms were obtained in all patients. Whole-body bone images were obtained with a scintillation camera fitted with a Div/Con collimator, with the diverging side towards the patient, 2–3 hr after intravenous injection of 15 mCi of $^{99m}$Tc-PP$_1$. Anterior and lateral views of the skull and mandible, and anterior views of the feet and hands were obtained; 300,000 counts were accumulated for each view. The bone images were subjectively graded: 1+ being equal to normal uptake; 2+, slightly increased; and 3+, markedly increased in comparison with the normal adjacent bone or the contralateral normal site. A grade of zero indicated decreased uptake. Bone uptake intensity was visually compared to the kidney intensity.

Blood and urine samples were collected as described elsewhere (10–12). Plasma clearance of $^{99m}$Tc-PP$_1$ was calculated by means of the standard clearance formula. Urine was collected hourly for 4 hr. For calculation of clearance during the first hour, the plasma count at 30 min was used. For calculation of clearance at the end of the 2nd, 3rd, and 4th hours, the mean plasma counts at the beginning and end of the hour were used.

Bone roentgenograms and scintillation camera images were interpreted independently by the radiologists and the authors; the readers were informed of the possible clinical diagnosis of metabolic bone disease. Such specific skeletal abnormalities as cyst and sub-periosteal resorption and such nonspecific changes as diffuse demineralization were sought for (Table 1). The control group consisted of six patients with histologically proven malignant soft-tissue tumors and with normal serum levels of calcium, phosphorus, and alkaline phosphatase. The serum parathormone levels were not measured in these patients, but there was no clinical or biochemical evidence to suspect any parathyroid disease. All had normal renal function bone images, and there was no uptake of $^{99m}$Tc-PP$_1$ by the primary tumor. The blood disappearance and urinary excretion of $^{99m}$Tc-PP$_1$ were also compared with four other patients, who resembled the control group of patients in every respect but who in addition had metastatic lesions in the bone. Blood and urine clearance values were tested by Student's t-test for statistical significance.

RESULTS

The clinical, biochemical, radiologic, and histologic findings in each patient are shown in Table 1. Eleven of 12 patients with primary hyperparathyroidism had hypercalcemia and most of them had hypophosphatemia with an elevation of serum PTH. Of the seven patients who underwent parathyroid surgery, four had single adenomas and three had parathyroid hyperplasia. Five of the patients had definite elevations of alkaline phosphatase and two were at the upper limits of normal. Five patients showed generalized demineralization of bone, one patient had subperiosteal resorption in the metacarpals, while the remaining six patients had normal bone radiologic studies. In seven patients, focal bone lesions were shown by the scintillation camera images, and five of these patients had no corresponding lesions by radiographic studies. The majority of lesions were confined to distal parts of the extremities, skull, and jaw bones (Table 1).

In five of 11 hyperparathyroid patients, bone mineral content in the distal third of the radius was reduced compared with age- and sex-matched controls.
### TABLE 1. BIOCHEMICAL, RADIOLOGIC, HISTOLOGIC, BONE MINERAL, AND BONE IMAGE CHANGES IN PARATHYROID DISEASE

<table>
<thead>
<tr>
<th>Pt. No.</th>
<th>Age/ Sex/Race</th>
<th>Calcium (mg/dl)</th>
<th>Phosphorus (mg/dl)</th>
<th>Alkaline phosphatase (30-85) (mg/ml)</th>
<th>Bone calcium (distal radius) (gm/cm²)</th>
<th>Radiographs</th>
<th>Radionuclide images</th>
<th>Surgical findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>PRIMARY HYPERPARATHYROIDISM</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>1</td>
<td>53/M/C</td>
<td>11.60</td>
<td>2.0</td>
<td>1.75</td>
<td>180</td>
<td>65</td>
<td>0.75(N)</td>
<td>Subperiosteal resorption-Demineralization</td>
</tr>
<tr>
<td>2</td>
<td>55/M/C</td>
<td>11.80</td>
<td>2.5</td>
<td>0.83</td>
<td>85</td>
<td>55</td>
<td>0.70(L)</td>
<td>Normal</td>
</tr>
<tr>
<td>3</td>
<td>51/F/C</td>
<td>12.50</td>
<td>2.6</td>
<td>0.40</td>
<td>70</td>
<td>67</td>
<td>Normal</td>
<td>Abnormal, left radius ++++ M-P joint of rt thumb +++</td>
</tr>
<tr>
<td>4</td>
<td>47/M/C</td>
<td>11.90</td>
<td>4.0</td>
<td>0.76</td>
<td>70</td>
<td>86</td>
<td>0.89(N)</td>
<td>Demineralization</td>
</tr>
<tr>
<td>5</td>
<td>58/M/C</td>
<td>10.80</td>
<td>3.4</td>
<td>0.62</td>
<td>58</td>
<td>88</td>
<td>0.78(L)</td>
<td>Demineralization</td>
</tr>
<tr>
<td>6</td>
<td>34/M/C</td>
<td>10.70</td>
<td>3.6</td>
<td>0.35</td>
<td>80</td>
<td>80</td>
<td>0.85(N)</td>
<td>Normal</td>
</tr>
<tr>
<td>7</td>
<td>25/M/C</td>
<td>10.00</td>
<td>2.7</td>
<td>0.70</td>
<td>65</td>
<td>84</td>
<td>0.85(N)</td>
<td>Normal</td>
</tr>
<tr>
<td>8</td>
<td>69/M/B</td>
<td>10.70</td>
<td>3.6</td>
<td>0.86</td>
<td>90</td>
<td>82</td>
<td>0.72(N)</td>
<td>Normal</td>
</tr>
<tr>
<td>9</td>
<td>43/M/C</td>
<td>11.50</td>
<td>2.0</td>
<td>0.60</td>
<td>95</td>
<td></td>
<td>0.77(N)</td>
<td>Normal</td>
</tr>
<tr>
<td>10</td>
<td>47/F/B</td>
<td>12.40</td>
<td>3.0</td>
<td>—</td>
<td>105</td>
<td>54</td>
<td>0.54(L)</td>
<td>Normal</td>
</tr>
<tr>
<td>11</td>
<td>52/F/C</td>
<td>11.20</td>
<td>2.9</td>
<td>0.70</td>
<td>120</td>
<td></td>
<td>0.55(N)</td>
<td>Normal</td>
</tr>
<tr>
<td>12</td>
<td>61/F/C</td>
<td>11.40</td>
<td>3.0</td>
<td>0.42</td>
<td>85</td>
<td>74</td>
<td>0.45(L)</td>
<td>Demineralization</td>
</tr>
</tbody>
</table>

### PSEUDOHYPOPARATHYROIDISM

<table>
<thead>
<tr>
<th>Pt. No.</th>
<th>Age/ Sex/Race</th>
<th>Calcium (mg/dl)</th>
<th>Phosphorus (mg/dl)</th>
<th>Alkaline phosphatase (30-85) (mg/ml)</th>
<th>Bone calcium (distal radius) (gm/cm²)</th>
<th>Radiographs</th>
<th>Radionuclide images</th>
<th>Surgical findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>35/M/C</td>
<td>6.0</td>
<td>4.5</td>
<td>0.95</td>
<td>160</td>
<td>91</td>
<td>0.82(N)</td>
<td>Short metacarpal bones</td>
</tr>
<tr>
<td>2</td>
<td>34/F/C</td>
<td>7.9</td>
<td>4.0</td>
<td>0.55</td>
<td>70</td>
<td>92</td>
<td>0.73(N)</td>
<td>Demineralization</td>
</tr>
<tr>
<td>3</td>
<td>27/F/C</td>
<td>8.5</td>
<td>4.2</td>
<td>0.45</td>
<td>70</td>
<td>89</td>
<td>0.71(N)</td>
<td>Normal</td>
</tr>
<tr>
<td>4</td>
<td>36/F/C</td>
<td>7.9</td>
<td>5.0</td>
<td>0.63</td>
<td>105</td>
<td>87</td>
<td>0.58(L)</td>
<td>Short metacarpal bones</td>
</tr>
<tr>
<td>5</td>
<td>16/F/C</td>
<td>9.0</td>
<td>4.5</td>
<td>0.41</td>
<td>55</td>
<td>93</td>
<td>0.53(L)</td>
<td>Short metacarpal bones</td>
</tr>
</tbody>
</table>

### HYPOPARATHYROIDISM

<table>
<thead>
<tr>
<th>Pt. No.</th>
<th>Age/ Sex/Race</th>
<th>Calcium (mg/dl)</th>
<th>Phosphorus (mg/dl)</th>
<th>Alkaline phosphatase (30-85) (mg/ml)</th>
<th>Bone calcium (distal radius) (gm/cm²)</th>
<th>Radiographs</th>
<th>Radionuclide images</th>
<th>Surgical findings</th>
</tr>
</thead>
<tbody>
<tr>
<td>1</td>
<td>45/M/B</td>
<td>6.5</td>
<td>5.1</td>
<td>Undetectable</td>
<td>40</td>
<td>91</td>
<td>0.52(L)</td>
<td>Normal</td>
</tr>
<tr>
<td>2</td>
<td>55/F/B</td>
<td>7.5</td>
<td>8.4</td>
<td>Undetectable</td>
<td>35</td>
<td>87</td>
<td>0.40(L)</td>
<td>Normal</td>
</tr>
<tr>
<td>3</td>
<td>51/M/C</td>
<td>6.0</td>
<td>4.9</td>
<td>Undetectable</td>
<td>55</td>
<td>90</td>
<td>0.69(N)</td>
<td>Normal</td>
</tr>
</tbody>
</table>

C = Caucasian  
B = Black  
PTH = parathormone  
TRP = Tubular reabsorption of phosphorus  
L = Low  
N = Normal bone mineral for age- and sex-matched controls  
* Serum PTH levels were measured through the courtesy of W. Fred Singer, The University of Southern California School of Medicine.
All five patients with pseudohypoparathyroidism had elevated PTH levels, and four had hypocalcemia. The fifth patient was normocalcemic but had an abnormal response to parathyroid extract. In two of these patients, alkaline phosphatase levels were elevated. Two patients had radiologic evidence of brachydactyly and one had generalized demineralization. Bone image abnormalities in four of five patients were found mainly in the hands at the periarticular regions.

The three patients with hypoparathyroidism had hypocalcemia at the time of the study. Bone radiologic studies were normal and only one patient showed bone image abnormalities. The lesions were confined primarily to the mandible and the periarticular regions of the big toe and thumb.

Blood radioactivity disappearance curves following intravenous injection of $^{99m}$Tc-PP$_1$ were similar for all groups of patients (Fig. 1). The urinary excretion of $^{99m}$Tc-PP$_1$, however, was relatively increased in those patients with primary hyperparathyroidism and pseudohypoparathyroidism compared with the controls (normal bone) (Fig. 2), and it was significantly increased compared to the four patients with metastatic bone disease ($p < 0.05$).

The plasma clearance of $^{99m}$Tc-PP$_1$ was relatively increased in patients with primary hyperparathyroidism in comparison with the control patients. The difference was more obvious during the first hour: $54.7 \pm 11.6 \text{ ml/min}$ in primary hyperparathyroidism versus $38.6 \pm 9.6 \text{ ml/min}$ in control subjects. The differences between the two groups, however, were not statistically significant (Table 2).

**DISCUSSION**

The original description of primary hyperparathyroidism emphasized the presence of skeletal abnormalities. With the recognition of the broad clinical spectrum of this disorder, the incidence of roentgenographically recognizable bone disease associated with the biochemical manifestations of hyperparathyroidism has diminished. This has been, in large part, related to the insensitivity of the techniques employed to evaluate bony abnormalities. Thus, in three series of patients with primary hyperparathyroidism, the incidence of x-ray abnormalities attributable to hyperparathyroidism ranged from 10 to 33% (6,13,14). The significance of the finding of osteopenia in patients with primary hyperparathyroidism is even less clear. Other workers have emphasized the

**TABLE 2. TECHNETIUM-99m-PYROPHOSPHATE CLEARANCE (ml/min) FROM PLASMA AFTER A SINGLE INJECTION (mean ± s.e.m.)**

<table>
<thead>
<tr>
<th>Time after injection</th>
<th>Primary hyperparathyroidism ($n = 9$)</th>
<th>Control ($n = 5$)</th>
</tr>
</thead>
<tbody>
<tr>
<td>0–1 hr</td>
<td>$54.7 \pm 11.6$</td>
<td>$38.6 \pm 9.6$</td>
</tr>
<tr>
<td>1–2 hr</td>
<td>$40.9 \pm 5.1$</td>
<td></td>
</tr>
<tr>
<td>2–3 hr</td>
<td>$26.1 \pm 3.2$</td>
<td></td>
</tr>
<tr>
<td>3–4 hr</td>
<td>$23.2 \pm 6.1$</td>
<td></td>
</tr>
</tbody>
</table>

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subjective nature of this radiologic finding (14). More recently, the use of bone mineral analysis employing photon densitometry has revealed varying degrees of skeletal mineral abnormality in patients with primary hyperparathyroidism. One study revealed an incidence of 50% (14), while in our small series four of eleven patients (36%) had low bone mineral. Finally, the incidence of histologic abnormalities on bone biopsy which is considered consistent with excess parathyroid hormone activity is much greater than the bone lesions detected by roentgenography, or than the measurement of serum alkaline phosphatase. In more than 90% of patients with proven or suspected hyperparathyroidism, abnormal findings have been reported by several investigators employing a combination of qualitative techniques or quantitative histologic methods (13, 15–17).

In the present studies, only one of 12 patients with primary hyperparathyroidism had evidence of increased subperiosteal resorption by routine radiographs. This patient had the highest alkaline phosphatase value of the group. In contrast, seven of the remaining patients had distinctly abnormal findings on bone imaging with 90mTc-PPi. Five of these patients had alkaline phosphatase values in the normal range, and four had normal values for bone mineral content. In the entire group of 12 patients, 10 had evidence of skeletal disease by radiologic examination, bone densitometry, or skeletal imaging. The latter technique appeared to be the most sensitive for this group of patients. Figure 3A shows a normal radiograph of the wrist and hand of a patient with primary hyperparathyroidism: Fig. 3B showed focal lesions in the radionuclide image of the left radius and the right metacarpal bones of the same patient.

Four of the five patients with pseudohypparathyroidism and one of the three patients with hypoparathyroidism showed skeletal abnormalities by radionuclide imaging. Only two patients with pseudohypparathyroidism, however, and no patients with hypoparathyroidism had roentgenographically demonstrable lesions. The bone mineral content was reduced in three of five patients with pseudohypoparathyroidism and one of three patients with hypoparathyroidism. Even in this small group of patients bone imaging detected more lesions than the other two diagnostic tests.

Most of the skeletal abnormalities detected in the radionuclide studies in these patients with parathyroid disorders were located in the peripheral parts of the body: the distal areas of both the lower and upper extremities, mandible, and the bones of the skull. Similar patterns of distribution were reported by two other groups of investigators (18,19). The skeletal distribution pattern of lesions in parathyroid disorders differs from that of metastatic bone disease where the lesions are more often found in the axial skeleton, especially the vertebral bodies, pelvis, and the proximal bones of the upper and lower extremities. The peripheral distribution of bone lesions in parathyroid disorders also differs from the distribution of lesions in patients with rheumatoid, psoriatic, and gouty arthritis in that it is focal and asymmetric (20). In arthritis the concentration of radioactive tracer is due mainly to the inflamed synovial membrane in the involved joints (21). Some patients with parathyroid disease, however, may show periarticular uptake resembling the changes seen in arthritis (Fig. 4). Generalized increased uptake is often observed in patients with secondary hyperparathyroidism, but this feature was not commonly seen in our patients with primary hyperparathyroidism. Focal abnormality was the predominant feature.

Technetium-99m-pyrophosphate is avidly taken up by both organic and mineral bone matrix, especially by the immature collagen of the organic phase during bone formation (21). Local vascularity also plays a significant role. Parathormone has varied biologic effects on bone in man; increasing activation of osteoprogenator cells; inhibiting modulation
of osteoclasts to osteoblasts; increasing the activity of pre-existing osteoclasts; and inhibiting pre-existing osteoblastic activity.

After a prolonged continuous increase in serum parathormone a secondary rise in the osteoblast pool often occurs (22). This secondary rise in the osteoblast pool, however, usually fails to maintain normal mineral balance and the skeleton enters into a negative balance. As reflected by increased urinary excretion of hydroxyproline (21), both animals and humans with hyperparathyroidism, rickets, osteomalacia, and Paget's disease have increased collagen turnover. This new collagen has a special affinity for \(^{99m}\)Tc-PP\(_i\), which results in "hot" spots in bone imaging studies. The appearance of "hot" spots in one of the three patients with hyperparathyroidism was an unexpected result. The overall rate of skeletal remodeling is very much decreased in hyperparathyroidism. Untreated chronic hyperparathyroidism patients manifest a small net increase in bone formation over bone resorption at the endosteal surface due to modulation of osteoclasts to osteoblasts (22). This slight increase in osteoblastic activity may explain the increased radioactive tracer uptake noted in the patient with hyperparathyroidism (Table 1).

Blood disappearance curves following injection of \(^{99m}\)Tc-PP\(_i\) were similar for all groups of patients (Fig. 1). A tendency towards increased urinary excretion of \(^{99m}\)Tc-PP\(_i\), however, was seen in patients with primary hyperparathyroidism and pseudohypoparathyroidism, all of whom had raised levels of serum parathormone (Fig. 2).

The plasma clearance rate of \(^{99m}\)Tc-PP\(_i\) was relatively increased during the first 2 hr after intravenous administration in patients with primary hyperparathyroidism in comparison to the controls. The difference was most apparent during the first hour (Table 2). Since blood radioactivity levels were identical for primary hyperparathyroid and control patients, the increased plasma clearance can be attributed to increased urine excretion (Fig. 2). This may be appreciated better by comparing the kinetic data of the hyperparathyroid patients with the patients who had metastases. The increased urinary excretion in patients with hyperparathyroidism can be attributed primarily to increased parathormone levels, in the metastatic group of patients there was no reason to suspect an abnormal parathormone levels.

The increased urinary excretion of \(^{99m}\)Tc-PP\(_i\), found in patients with pseudohypoparathyroidism is difficult to interpret. Plasma clearance was not measured in these patients. Classically, these patients are known to have end organ resistance to PTH and one would anticipate a normal or decreased urinary excretion of phosphates to PTH infusion. The addition of tin to the pyrophosphate complex may have played a role in altering the excretion pattern of the \(^{99m}\)Tc-Sn-pyrophosphate. The excretion of \(^{99m}\)Tc-PP\(_i\), in patients with hyperparathyroidism and controls was similar. One may infer therefore that the elevated serum parathormone will increase urinary excretion of \(^{99m}\)Tc-PP\(_i\).

ACKNOWLEDGMENT

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REFERENCES


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**Pamphlet #1, Revised**—A Revised Schema for Calculating the Absorbed Dose from Biologically Distributed Radionuclides—12 pp.

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