INCREASED ^{99m}Tc-POLYPHOSPHATE MUSCLE UPTAKE IN A PATIENT WITH POLYMYOSITIS: CASE REPORT

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A patient with well-documented rheumatoid arthritis and polymyositis displayed abnormal muscle uptake of ^{99m}Tc-polyphosphate during routine bone scanning for occult malignancy. The regions of increased uptake corresponded to the areas of clinically active inflammatory muscle disease. On serial scans the degree of muscle labeling correlated well with both clinical and laboratory indices of disease activity.

This paper reports an instance of increased muscle labeling in a patient with well-documented clinically active polymyositis. Serial ^{99m}Tc-polyphosphate bone scans showed a correlation between the degree of muscle labeling and the activity of the inflammatory myopathy.

Although the incidence of malignancy in patients with polymyositis is probably not as high as previously reported (*1–3*), there are good grounds for believing that the incidence is higher than in the general population. The increased suspicion of malignancy in these patients often leads to a search for occult neoplasm. A bone scan done for this purpose on a patient with polymyositis showed abnormalities in muscle labeling which are probably attributable to the inflammatory myopathy. Two subsequent scans, reviewed in light of the clinical course, supported this conclusion.

CASE REPORT

A 39-year-old black woman was admitted to the hospital with complaints of arthralgias, proximal muscle weakness, and pain in the anterior thigh muscles. Three years previously she had first noted the onset of pain and swelling of both knees and wrists, for which she was treated with phenylbutazone with minimal improvement. Corticosteroid therapy had been initiated 1 year prior to admission. Physical examination at the time of admission revealed questionable synovial thickening of both wrists and pain on compression of the proximal interphalangeal joints. There were no significant joint deformities. Neurologic examination revealed severe proximal muscle weakness with tenderness to palpation of the quadriceps femoris bilaterally. Laboratory tests showed a hemoglobin of 12.9 gm/100 ml and a white blood count of 15,000. The erythrocyte sedimentation rate was elevated at 44 mm/hr. A rheumatoid-factor determination was positive. The serum lactic dehydrogenase (LDH) was 775 mU/ml (normal $<$ 220), serum glutamic oxaloacetic transaminase (SGOT) 500 mU/ml (normal $<$ 60), and serum creatinine phosphokinase (CPK) 3,590 mU/ml (normal $<$ 70). Before increasing the patient's steroid dosage, it was necessary to rule out the possibility of a steroid-induced myopathy. A muscle biopsy of the right quadriceps was performed and interpreted as showing severe inflammatory myopathy with perifascicular atrophy, suggesting polymyositis. No abnormal calcification was noted. The patient was discharged with a diagnosis of rheumatoid arthritis and polymyositis; the discharge medication included 60 mg/day of prednisone.

Following discharge the patient's weakness and muscle pain improved and serum enzyme values returned to normal. She continued to do well until 2 months after her first admission, when she had an exacerbation of quadriceps weakness and was readmitted to the hospital. Physical examination again revealed proximal muscle weakness and tenderness. The serum enzyme values were again elevated, with an LDH of 1,390 and an SGOT of 1,575. To rule out an hepatic origin for the elevated enzymes, a percutaneous liver biopsy was performed, the results of which were normal. The illness was ascribed to an exacerbation of myositis. A whole-body bone scan

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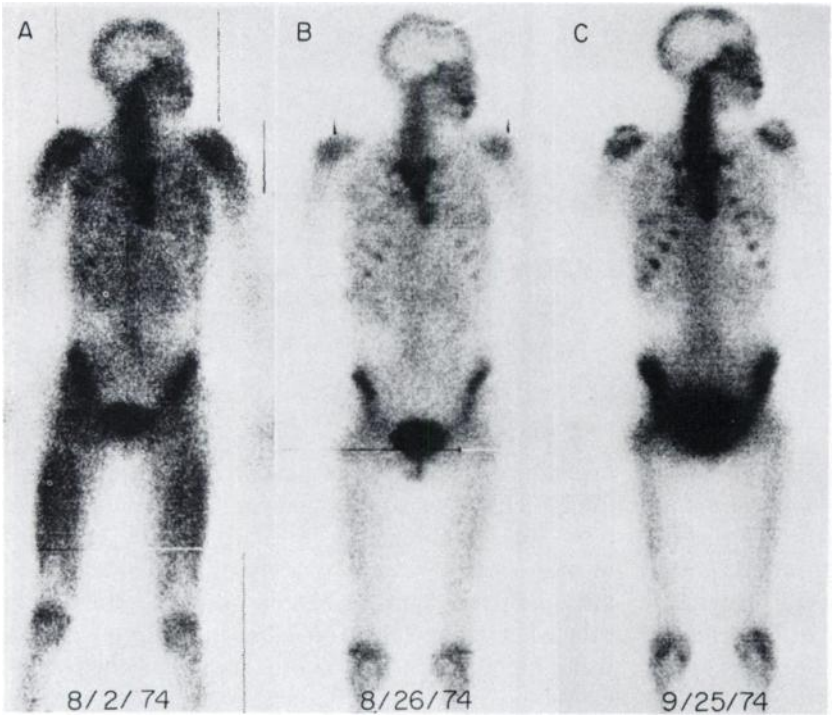

FIG. 1. Anterior ^{99m}Tc-polyphosphate bone scans of patient with well-documented polymyositis. (A) Scan obtained during acute phase of illness shows increased labeling of proximal musculature of arms and thighs. There was severe weakness and tenderness of these muscles and serum enzymes were markedly elevated. (B) Scan after 1 month of high-dose steroid therapy shows interval reduction in muscle labeling. Weakness and muscle tenderness were much improved, and serum enzymes were within normal limits. (C) Scan after 2 months of therapy similar to (B) but with further reduction in thigh labeling. Although some residual weakness remained, muscle tenderness had cleared. Serum enzymes were normal.

was performed on the fifth hospital day, using New England Nuclear ^{99m}Tc-Sn-polyphosphate and an Ohio-Nuclear dual-probe scanner (Fig. 1A). The scan showed increased labeling in the shoulders and knees consistent with the known arthropathy. In addition, soft-tissue uptake in the proximal third of both arms and in the muscle groups of both thighs was increased. The prednisone dose was increased to 100 mg/day and the muscle weakness and tenderness receded. At the same time, serum enzyme values fell toward normal (LDH 427, SGOT 197). The patient was discharged.

Three weeks after discharge the patient's muscle weakness and tenderness were found improved. Laboratory results for enzymes were normal. A repeat bone scan (Fig. 1B) showed increased uptake in the large joints as before. The abnormal uptake in the proximal arms and in the thighs, although still present, was much less dramatic.

TABLE 1. CORRELATION OF CLINICAL, LABORATORY, AND SCAN DATA

	8/2/74	8/24/74	9/25/74
Muscle weakness	Severe	Moderate	None
Tenderness	Severe	Moderate	None
SGOT (n < 60)	1,575	197	38
LDH (n < 220)	1,390	427	255
CPK (n < 70)	3,590	26	—
Muscle uptake on scan	Marked	Slight	Normal

The patient continued to improve on high-dose steroid therapy but was again admitted 4 months after her initial hospitalization for control of steroid-induced glucose intolerance. At this time, subjective and objective indications of muscle disease were unchanged. A third whole-body scan performed during this admission showed (Fig. 1C) findings similar to the second study, with slight further decrease in thigh uptake.

DISCUSSION

The introduction of the ^{99m}Tc-labeled phosphate compounds has had a significant impact on whole-body bone scanning (*4*). Shortly after these agents attained widespread clinical use, reports of abnormal accumulations in nonosseous structures began to appear in the literature (*5*). Of particular interest are recent reports of ^{99m}Tc-polyphosphate uptake in acutely infarcted myocardium (*6–8*).

We have presented a case of polymyositis with typical clinical, laboratory, and histologic findings as judged by the criteria recently proposed by Bohan and Peter (*1,9*). In this patient, abnormal uptake of ^{99m}Tc-polyphosphate in clinically affected muscle groups appeared in routine bone scanning. Furthermore, following successful treatment with corticosteroids, subsequent scans correlated with clinical and laboratory evidence of improvement (Table 1).

The mechanism for the increased muscle uptake is not known. It has been shown in animal models that the affinity of normal muscle for ^{99m}Tc-poly-

phosphate is quite low compared to that of bone, kidney, spleen, blood, liver, and lung (*10*). Perhaps the damaged muscle tissue has an increased avidity for calcium, like that accounting for the labeling of recent myocardial infarctions with ^{99m}Tc-polyphosphate (*8*). The muscle biopsy in our patient did not reveal any abnormal calcification. However, abnormal calcification has been shown by electron microscopy in patients with muscle damage (*11*). Areas of greatest bulk of abnormal muscle would be expected to show the most prominent labeling, as in the present case.

We are currently examining ^{99m}Tc-polyphosphate bone scans in other patients with inflammatory muscle disease. The use of bone scans to evaluate these cases seems promising (*12,13*).

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