HIV-Associated Myositis Detected by Radionuclide Bone Scanning

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We report a case of myositis associated with human immunodeficiency virus (HIV) reactivity which was demonstrated on bone scan in the presence of normal muscle enzymes, contrast computed tomography, and electromyography. The diagnosis was confirmed by muscle biopsy. Radionuclide bone scanning may be a valuable diagnostic tool in HIV positive patients who present with muscle pain and an otherwise normal diagnostic evaluation.

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The first report of technetium-99m- (^{99m}Tc) labeled phosphate uptake in inflammatory muscle disorders was made by Spies et al. in 1975 (*1*). The finding of increase uptake in inflamed muscle has since been confirmed in several studies (*2–5*). We report a case in which the radionuclide bone scan represented the only diagnostic test suggesting the diagnosis of myositis in an HIV-positive individual presenting with severe bilateral muscle pain.

CASE REPORT

The patient was a 37-yr-old black male with a history of intravenous drug abuse. He presented with a 2½-year history of leg pains, predominantly on the right. Seventeen months prior to admission a bone scan (Fig. 1) was performed. The study was performed 2.5 hr after the intravenous injection of 840 MBq [^{99m}Tc]methylene diphosphate (MDP). Images were obtained using a large field of view gamma camera equipped with a high resolution parallel hole collimator. The scan revealed mildly increased soft-tissue uptake in the anterior thigh on the right. Contrast-enhanced computed tomograms of the pelvis and right thigh performed at that time were normal although an arteriogram revealed right popliteal artery occlusion, which was thought to be responsible for his complaints. The patient was found to have cryoglobulinemia. Assay for HIV antibody was positive. The patient experienced no further leg symptoms until 2 days prior to the current admission, when he developed bilateral leg pains. These were accentuated by motion and associated with a low grade fever (99.8°F). He reported a history of intermittent sweats over the preceding months. The thigh muscles were not swollen, however, tenderness was observed in the hamstrings > quadriceps > gastrocnemii. Creatine kinase and lactate dehydrogenase were normal on repeat measurements and pulses in the left foot were within normal limits. The remainder of the physical examination was normal except for a 1-cm supraclavicular lymph node. The erythrocyte sedimentation rate was markedly elevated at 126 mm/hr. Alkaline phosphatase was 90 U/l, serum glutamic oxaloacetic transaminase (SGOT) 46 U/l, angiotensin converting enzyme (ACE) normal, rheumatoid factor 162 IU/ml, antinuclear antibody (ANA) negative, fluorescent treponemal antibody (FTA) negative, rapid plasma reagin (RPR) positive and cryoglobulin negative on this admission (although positive in the past). The creatinine was slightly elevated at 1.6 mg/dl. The hematocrit and platelet count were both mildly decreased. Cerebrospinal fluid (CSF) was negative for cryptococcus and a venereal disease research laboratory test (VDRL) was negative. Plain film radiographs of the thigh were normal as were electromyography (EMG) results. A bone scan was performed 3 hr after the i.v. injection of 820 MBq [^{99m}Tc]MDP (Fig. 2), demonstrating increased activity in the muscle groups of the thighs bilaterally, left greater than right. The abnormality was most prominent in the hamstring group and in the adductors. Slightly asymmetric activity was shown in the inferior pubic rami, a finding felt to be of uncertain clinical significance. A contrast-enhanced CT of the thighs (Fig. 3) was interpreted as normal, although in retrospect there may be slight loss of definition of the muscle planes on the left. A muscle biopsy of the gastrocnemius showed interstitial myositis and perivascular chronic inflammation without evidence of vasculitis.

DISCUSSION

Inflammatory muscle disease has been reported in association with acquired immunodeficiency syndrome

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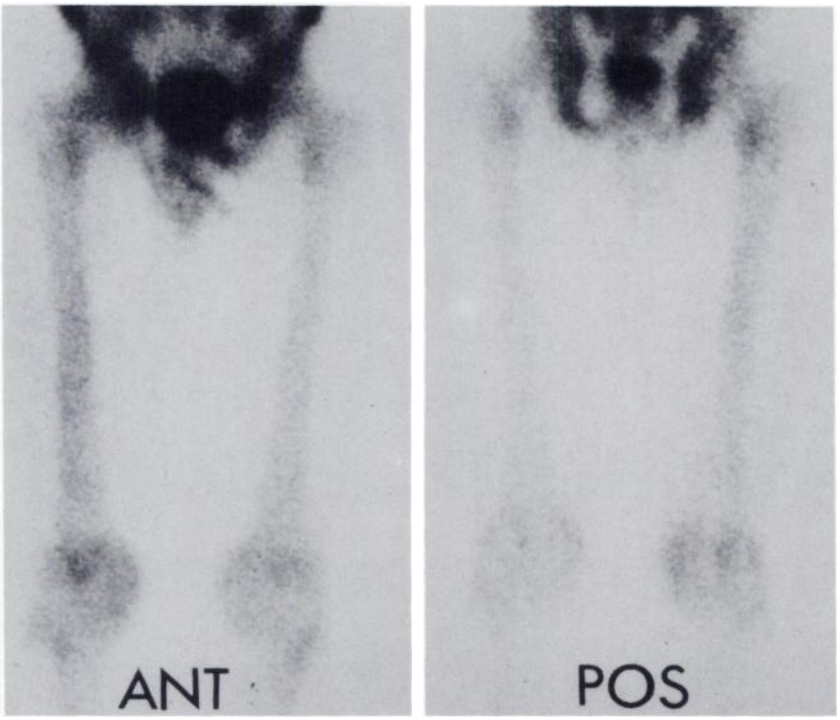

FIGURE 1
Bone scan performed 17 mo prior to admission showing a subtle increase in uptake in the right thigh.

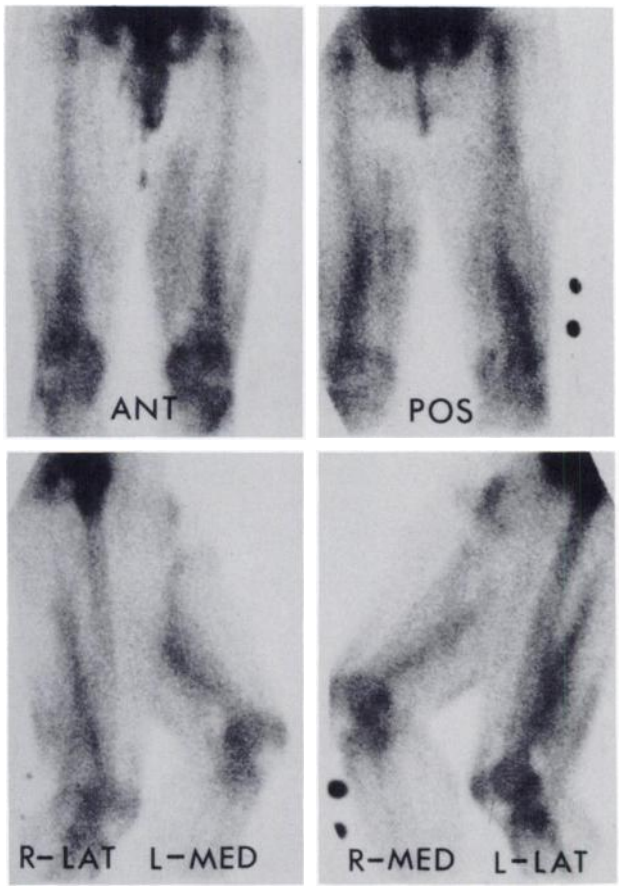

FIGURE 2
Bone scan performed at the time of admission showing markedly increased activity in the thighs bilaterally, left greater than right and most prominent in the hamstring and adductor groups.

(AIDS) (*6,7*) and may be more common than has previously been recognized. It is characterized by tenderness and weakness of the involved muscle groups, most pronounced in the distal lower extremities. Patients have been reported to have normal creatine kinase (CK) levels on repeated testing and the syndrome has a benign course. Some cases have shown multinucleated giant cells in affected tissue. The muscle disorder may present as the first symptom of an impending AIDS-related complex or as a full manifestation of AIDS.

The patient had evidence of cryoglobulinemia at the time of initial presentation but not at follow-up. Cryoglobulinemia is occasionally associated with a peripheral neuropathy, with pain and paresthesias occurring upon exposure to cold (*8*). Weakness and muscle atrophy may appear, most severe in the legs, and often asymmetric. The pathogenesis of the syndrome is probably vascular and both prednisone and chlorambucil have been used to induce remission. Since there was no evidence of cryoglobulinemia at the admission in question, it is unlikely that this factor was related to the diagnosis of myositis.

Uptake of ^{99m}Tc diphosphonates has been shown in a rat model to occur within 12–24 hr of onset of a necrotizing myopathy caused by the myotoxin notexin (*9*). Activity was demonstrated in muscle mitochondria, edema fluid, and inflammatory cells. Denervation alone, either acute or chronic, was not sufficient to produce uptake of the tracer nor was uptake seen in regenerating muscle. Technetium-99m diphosphonate uptake correlated with total muscle Ca^{++} during the

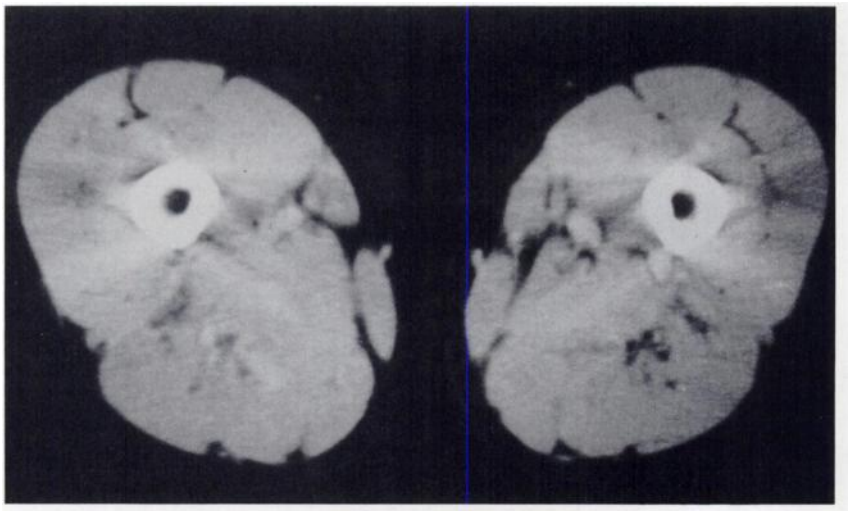

FIGURE 3
Contrast-enhanced CT scan of the thighs performed at the time of admission. This section was taken through the region of intensely increased activity on the bone scan. The study was interpreted as normal. In retrospect, there may be slight loss of definition in the muscle planes on the left.

initial 24 hr following onset of necrosis. These authors speculated that it was unlikely that the tracer would be able to cross an intact sarcolemma and suggested that it was the inflammatory component of the myopathy which was largely responsible for the accumulation of tracer in injured muscle. Other workers have confirmed these results in inflammatory myopathies and have shown a decrease in ^{99m}Tc pyrophosphate uptake after treatment with corticosteroids (*10*).

The presence of pyrophosphate uptake has proven useful in directing the site of biopsy in patients with suspected polymyositis and dermatomyositis, since focal inflammation may lead to negative biopsies in 10–15% of patients (*11,12*). Uptake has not correlated well with serum CK levels: patients with polymyositis and minimal CK elevation have shown marked tracer uptake while patients with Duchenne-type muscular dystrophy have shown minimal uptake in the presence of markedly elevated CK levels (*13*). Recently, SPECT quantitation of ^{99m}Tc pyrophosphate uptake has been shown to correlate linearly with the extent of muscle necrosis as quantitated by tetrazolium staining in a canine experimental model (*14*).

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