jnm/CASE REPORT

UPTAKE OF ^{99m}Tc BONE-SCANNING AGENT BY LUNGS WITH METASTATIC CALCIFICATION

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Uptake of ^{99m}Tc bone-scanning agent into lungs containing metastatic calcification was demonstrated in a patient with a carcinosarcoma of the bladder. This gives further support to the hypothesis that ^{99m}Tc bone agents are associated with hydroxyapatite.

Uptake of ^{99m}Tc-bone-scanning agents by extraosseous tissues has been frequently reported (*1–4*). However, concentration in an area of metastatic calcification has not, to our knowledge, been previously described. The present case is one of metastatic calcification associated with hypercalcemia accompanying malignant disease. The agent used was produced by the Australian Atomic Energy Commission and thought to be a technetium polyphosphate (*5*). Recent studies (*6*) have now shown this to be a mixture of pyrophosphate and orthophosphate as a result of hydrolysis accelerated by autoclaving.

CASE REPORT

The patient, a 52-year-old baker, was admitted to the hospital following an injury to his right hand. After surgical repair his mental state deteriorated. His speech became unintelligible and he was uncooperative. A metabolic cause was suspected. He was found to have hematuria and was in renal failure with a serum creatinine of 14.0 mg/100 ml and had severe hypercalcemia of 16.4 mg/100 ml. Serum parathyroid hormone assay was normal. Following rehydration and steroid therapy, the serum calcium level returned to normal with clinical improvement in the patient's mental state. Investigation of his renal tract revealed bilateral hydronephrosis and dilation of the ureters. A biopsy taken at cystoscopy was reported as a leiomyosarcoma. At subsequent surgery a right nephrostomy was performed. This was followed by temporary improvement, but the nephrostomy became infected and the patient's condition rapidly deteriorated and he died, probably with septicemia, 10 weeks after admission. A number of radionuclide studies was performed using a Toshiba, large-field, delay-line scintillation camera (GCA-202). A brain scan was normal. A renal scan showed the features of obstruction. A bone scan was performed and no evidence of skeletal metastases was found, which was in agreement with a radiologic survey that was also performed. The chest x-ray film was normal. Of special interest was a marked degree of uptake of bone compound in the thorax bilaterally. This was clearly not related to ribs or external structures and was presumably in the lungs (Fig. 1). The degree of uptake varied throughout the lungs, being most marked in the lower zones and on the right side.

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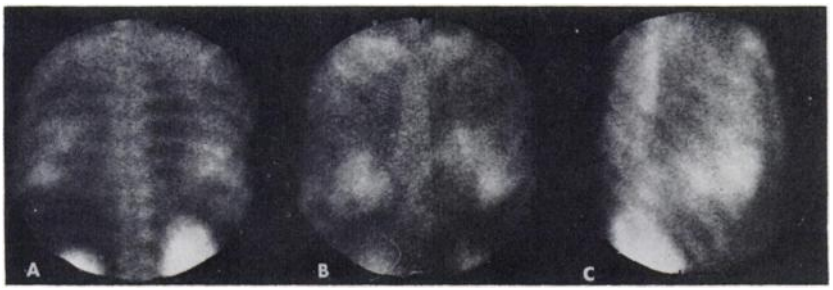

FIG. 1. Posterior (A), anterior (B), and right lateral (C) images of thorax showing bilateral accumulation of activity maximal at lung bases. (All images contain 900K counts.)

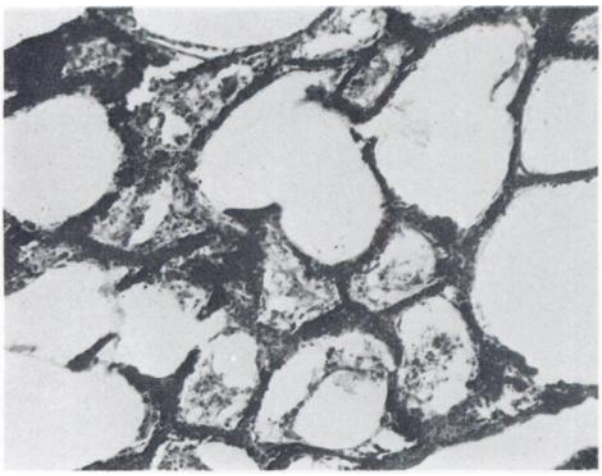

FIG. 2. Metastatic calcification in lung. Photomicrograph showing alveoli outlined by heavy calcification in walls. (Von Kossa stain × 250 prior to reduction.)

At autopsy the patient had peritonitis and an extensive carcinosarcoma involving both the bladder and a large associated diverticulum. There were bilateral hydroureters and hydronephrosis with pyelonephritis. Tumor metastases were present in the liver, mesentery, and spleen. There were no metastases in the lungs. Parts of the lungs, however, were pale and had a peculiar crisp texture. These areas corresponded exactly to the areas of abnormal isotope uptake. Histology showed calcification outlining the alveolar walls and in the walls of large vessels in the lung (Fig. 2). Calcification of lesser degree was also noted in the kidneys, bladder wall, and intramural vessels of the heart. Sections from bone showed only a mild increase in osteoclastic activity. It was presumed that the metastatic calcification was due to a hypercalcemic factor secreted by the bladder tumor.

DISCUSSION

Yeates, et al (*1*) have described the uptake of this bone agent into a parotid tumor and in another patient into presumed cerebral metastases from a parotid tumor. Berg, et al (*2*) described two patients in whom technetium diphosphonate accumulated in primary breast carcinoma. Uptake of technetium polyphosphate into an area of cerebral infarction has been described in a number of patients (*3,4*).

The mechanism of uptake by bone of technetium radiopharmaceuticals is thought to be adsorption onto the hydroxyapatite crystal (*6*). The same mechanism is probably responsible for uptake in areas of pathologic calcification that have been shown by x-ray diffraction studies to be similar in composition to normal calcification in bone (*7*).

This case was reported as an addition to the growing number of cases involving extraosseous uptake of ^{99m}Tc bone-seeking radiopharmaceuticals. It was also reported as further evidence that the mechanism of bone uptake is an affinity for hydroxyapatite crystals probably by ion exchange at their surface.

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