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Rhabdomyosarcoma Concentrating Tc-99m MDP

Extrasosseous tissue uptake of bone-seeking agents has been observed in many malignant and benign conditions. This nonspecific phenomenon has not been found due to a single mechanism, and many causes must be taken into account. Cases of uptake by various neoplasms, such as ovarian (1), liver (2), breast (3), and other primary and metastatic carcinomas (4,5) have been reported.

In soft-tissue sarcomas of adults and in neuroblastomas in children, the uptake of bone-seeking agents by the neoplasm is not an uncommon event and could probably play a clinical role (6). Howman-Giles et al. (7) have even suggested that neoplastic uptake of Tc-99m phosphate compounds in a pediatric patient would be almost pathognomonic of a neural-crest tumor. However, Magill and Strang (8) reported a case of a paraspinal isolated metastasis from a Wilms' tumor taking up Tc-99m MDP, and suggested the nonspecificity of this finding.

Accordingly, we present here a similar case of rhabdomyosarcoma in support of this statement.

A 5-yr-old boy was admitted to a pediatric ward because of the presence of a paravertebral spindle-shaped mass. The mass was observed after a right lumbar contusion. The child complained continuously about a "left-sided" lumbar pain. A chest radiograph confirmed the presence of a paravertebral mass. Hematochemical data showed increased levels of CPK (382 U/l) and LDH (305 U/l). The level of urinary catecholamines was within normal range. Bone imaging with Tc-99m MDP was done for clinical staging. It showed uptake in a paravertebral mass in both anterior

and posterior views (Fig. 1, left). The mass seemed to increase activity of the left tenth rib. No hyperactivity was observed in other skeletal structures. The radiological survey showed no bone abnormalities. A needle biopsy of the mass was performed and the histological diagnosis was "embryonic rhabdomyosarcoma" with dispersed infiltration of the skeletal muscle and diffuse necrosis.

The patient was treated according to AIEIP RMS 79 schedule: endoxan, vincristine, and actinomycin-D, combined with radiotherapy (44 Gy), with full radiological remission of the paravertebral mass. Repeat bone images performed at 6 mo after the end of the therapy showed no evidence of extrasosseous uptake (Fig. 1, right).

Many factors have been suggested to account for soft-tissue tumor uptake: (a) tumor neovascularity with altered capillary permeability (9); (b) binding of phosphate compounds by mitochondria in damaged (particularly necrotic) tissue (10); (c) binding of the radiopharmaceutical to soluble proteins resulting from denatured macromolecules (11). In our case it may be that the second mechanism was the most important cause of the uptake.

This case is a further demonstration of the nonspecificity of soft-tissue tumor uptake in pediatric patients.

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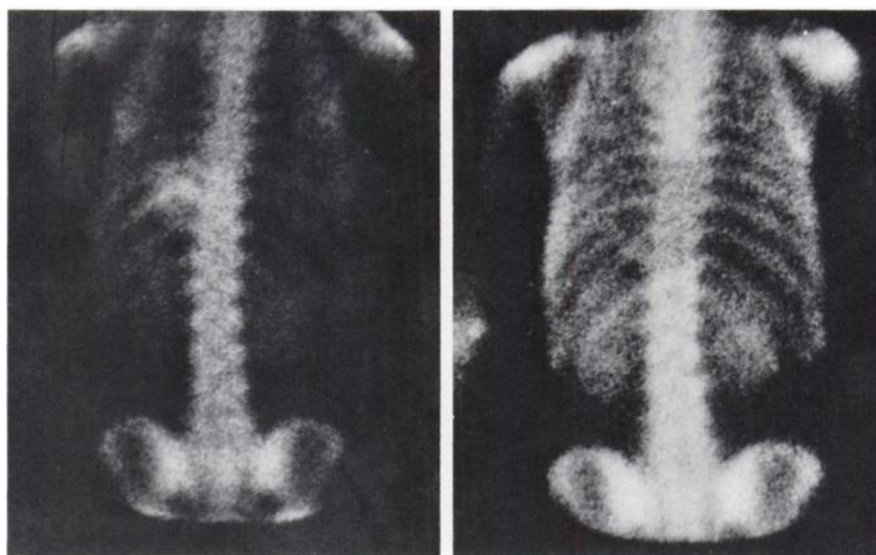


FIG. 1. Posterior bone image showing uptake in paravertebral mass (left): Bone study performed 6 mo after radiochemotherapy demonstrating no evidence of mass (right).

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Diffuse Lung Uptake of Tc-99m Sulfur Colloid in Infectious Mononucleosis

Diffuse uptake of Tc-99m sulfur colloid is an unusual phenomenon that has been associated with a variety of processes including hepatic disease (1-3), malignancy (4), sepsis (1,2), Type 2 mucopolysaccharidoses (5), collagen vascular disease (1), hematologic disorders (1), liver transplant (6), spleen and bone-marrow transplant (4), amyloidosis (7), and antacid therapy (8). We present a case of marked diffuse lung uptake of Tc-99m sulfur colloid in a patient with infectious mononucleosis.

A 19-yr-old white man was seen for a 10-day course of sharp left upper quadrant abdominal pain, increasing fatigue, appetite loss, and sore throat. Physical examination revealed the spleen tip palpable 6 cm below the costal margin, with mild tenderness, enlarged lymph nodes in the cervical and inguinal chains, and mild enlargement of the pharyngeal tonsils, with erythema. The diagnosis of infectious mononucleosis was confirmed with a positive monospot and a characteristic peripheral blood smear. Chest radiographs were normal.

Because of concern that the abdominal pain may have been due to a splenic subcapsular hematoma, a Tc-99m sulfur colloid liver/spleen study was performed. This showed splenomegaly and marked diffuse pulmonary accumulation of sulfur colloid, the activity in the lungs being as intense as that in the liver (Fig. 1). No evidence of colloid shift in the form of increased bone-marrow or splenic activity was seen.

The quality of the radiopharmaceutical was verified by examining the studies of six other patients injected with the same sulfur colloid preparation for evidence of increased lung uptake. Four showed no lung uptake. Two with severe hepatocellular disease and marked colloid shift to the spleen and bone marrow showed minimal lung uptake; this association has been well described by Steinbach (3). Optimal colloid size and lack of macroaggregation of particles was verified before and after this study, using a hemocytometer with light microscopy. Aluminum ion concentration in the preparation was less than 10 $\mu\text{g}/\text{ml}$ by colorimetric assay. While it has been reported that aluminum ion concentrations as low as 1 $\mu\text{g}/\text{ml}$ can cause formation of a flocculent precipitate in the colloid preparation (9), the minimum detectable aluminum ion concentration with commercial colorimetric assay kits is 10 $\mu\text{g}/\text{ml}$.

After the quality of the radiopharmaceutical has been assured, possible causes of diffuse increased uptake of sulfur colloid in the lungs would include: (a) in vivo macroaggregation or flocculation

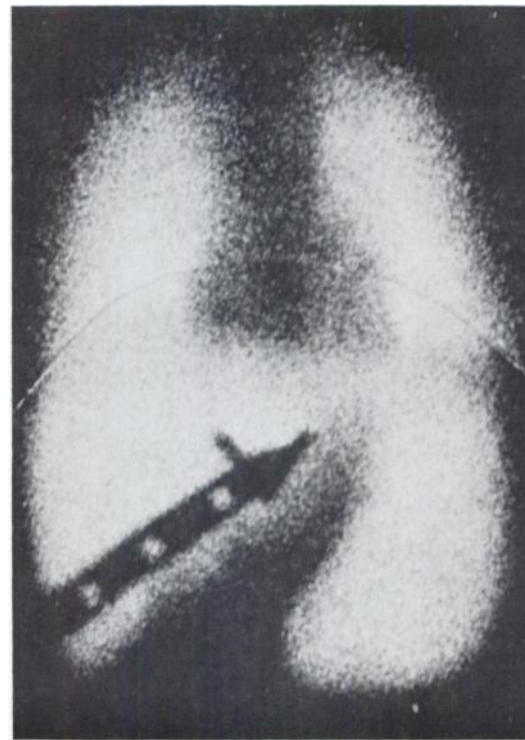


FIG. 1. Composite of identically exposed scintiphotos demonstrating marked diffuse lung uptake of Tc-99m sulfur colloid.

of particles, such as can be caused by excess plasma aluminum ion concentration (9) or by other as yet unknown factors; (b) adherence of the sulfur colloid particles to the endothelial cells of the pulmonary capillaries (1); or (c) increased phagocytic activity due to either increased activity of the small number of macrophages normally present in the pulmonary capillary bed or to an increased number of these macrophages (5). While we have no pathological proof at the present time, we believe that in a patient with infectious mononucleosis and an increase in the number of circulating blood monocytes, the last hypothesis would be most likely (5).

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