

Diffuse Increased Uptake on Bone Scan: Super Scan

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A 67-year-old woman who had suffered from agnogenic myeloid metaplasia (AMM) for 3 years was admitted with fever and a productive cough lasting 3 days. Physical examination re-

vealed coarse rales on the base of both lungs. Laboratory tests showed WBC 28,400/mm³ with a shift to the left, hemoglobin 11.5 mg/dL, and platelets 193,000/mm³. Biochemical findings showed Na 134 mm/L, K 4.9 mm/L, Ca 8.5 mg/dL, P 5.4 mg/dL, and normal kidney function. Chest X-ray showed enlarged heart with bilateral pleural effusion, larger on the right side, and slight lung infiltration in the right lower lobe. Diffuse symmetrical sclerotic densities throughout the thoracic cage bone, compatible with systemic bone marrow involvement, were also noted. Chest computed tomography examination showed widespread groundglass opacification of both lungs with huge splenomegaly and inhomogenously sclerotic tho-

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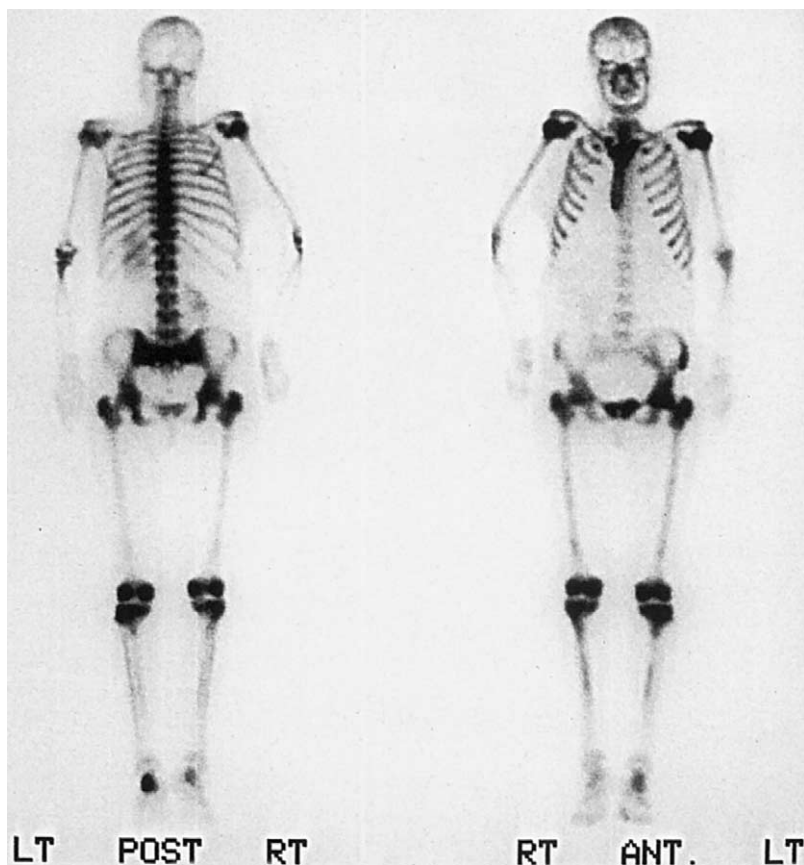


Fig 1. Whole body bone scintigraphy demonstrating diffusely increased activity in all bones, especially the joint area, with minimal activity in the kidney.

racic cage (not shown). Thereafter, bone scintigraphy was performed demonstrating diffusely increased activity in all bones, especially the joints, with minimal activity in the kidneys (Fig 1).

DISCUSSION

A super scan is defined as intense symmetric activity in the bones and diminished renal parenchymal activity. Such appearances have been reported in a variety of malignancies and metabolic bone disorders. It is well known that the uptake of a bone-seeking agent depends upon blood flow to the area and bone remodeling activity—in particular new bone formation.¹ Intense activity in bone scintigraphy in diffuse metastatic disease can be explained by diffuse bone marrow involvement. Metastases in bone are invariably found in sites adjacent to red bone marrow. This suggests that tumor cells lodge, survive, and grow in the hematopoietic bone marrow space and expand to destroy adjacent bone.² Frankel et al³ and Thrumkaew et al⁴ reported cases of diffuse metastatic disease of bones where bone scintigraphy showed a super scan. Sy et al⁵ hypothesized that the increased uptake of radiopharmaceutical by diseased bone results in reduced phosphate excretion, thereby producing faint renal images in the bone scintigraphy. Therefore, decreased visualization of the kidney in bone scintigraphy suggests the possibility of widespread bone disease. In metabolic bone disease, the exact mechanism of increased skeletal uptake is controversial. All patients with hyperparathyroidism have increased bone turnover on histomorphometry⁶ and would be expected to show increased bone pharmaceutical uptake. Osteomalacia, however, is associated with slow bone turnover.⁷ Rosenthal and Kaye suggested that Tc-99m MDP binds to immature collagen in osteomalacic bone.⁸ Another explanation proposes that there may be so much osteoid present that even though mineralization is occurring more slowly than normal at any given site, the total area of mineralization is increased.⁹ The mechanism involved in renal osteodystrophy is complex and there are numerous interactive factors, but the most important of them are osteomalacia and hyperparathyroidism. Bone turnover is also increased in

hyperthyroidism, but the increase in bone resorption is relatively greater than new bone formation. Longstanding hyperthyroidism may produce radiographic signs of osteoporosis.¹⁰ In AMM, there are some hypotheses regarding the mechanism of increased uptake. Fluorine-18 scintigraphic and kinetic data have shown a marked increase in skeletal blood flow in patients with myelofibrosis. In addition, bone resorption in cases of very advanced AMM causes the bone surface to increase many times. It has been proposed that these alterations could account for the increased tracer uptake of bone scintigraphy in myelofibrosis.¹¹ Early in the course of AMM, the marrow is often hypercellular, with maturing cells of all line ages being increased. During this cellular phase, fibrosis is minimal. With progression of the disease, the marrow becomes hypocellular and diffusely fibrotic. In the later stages of the disease, the fibrotic marrow space may be largely converted to bone, a development that is termed osteosclerosis.¹² It is of interest that many additional causes of super scan may accompany myelofibrosis such as renal osteodystrophy, vitamin D deficiency, hyperparathyroidism, carcinoma of the breast, lung, prostate, or stomach lymphoma, and leukemia.¹³ We believe that myelofibrosis may be one of the causes of super scan in these diseases.

Causes of Super Scan

Nonmalignant condition

Hyperparathyroidism¹⁴⁻¹⁷

Renal osteodystrophy⁷

Osteomalacia^{7,18}

Hyperthyroidism¹⁹

Acromegaly²⁰

Hypervitaminosis D^{20,21}

Mastocytosis²²

Malignant disorders:

Metastatic prostate carcinoma^{4,5,23-25}

Metastatic breast carcinoma^{4,26,27}

Metastatic lung carcinoma²⁷

Metastatic transitional cell carcinoma^{5,24,27}

Metastatic colon carcinoma²⁸

Lymphoma³

Agngenic myeloid metaplasia (myelofibrosis)^{29,30}

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