

firmed the presence of pseudofractures at some of the abnormal sites on the bone scan (Fig. 2A and 2B).

Pseudofractures are a recognized feature of osteomalacia and commonly occurs in the ribs (2). The patients described above were known to have severe osteomalacia and, although they complained of severe and persistent thoracic pains, the presence of pseudofractures was not suspected prior to bone scanning because of the generalized nature of these pains and the negative skeletal roentgenograms. There was no history of trauma to the thoracic cage in either patient. After the bone scan had revealed multiple "hot spots" over the ribs, coned roentgenograms of the areas of abnormality confirmed the presence of several pseudofractures, although these were still not seen in all the areas indicated by the bone scan.

From these two patients it appears that the radionuclide bone scan is a more sensitive means of detecting pseudofractures in the ribs than conventional radiology, even when coned roentgenograms are obtained. It also is important that adequate clinical information be available when interpreting such images since appearance is non-specific and could be misinterpreted as tumor metastases or other pathologic processes.

It is suggested that the bone scan should be part of the routine investigation of all patients with osteomalacia, because an awareness of the presence of pseudofractures is important in explaining the patient's symptoms. In addition, the physician may reassure the patient of a good prognosis, once treatment is commenced.

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### Simultaneous Occurrence of Functional Asplenia and Splenic Accumulation of Diphosphonate in Metastatic Breast Carcinoma

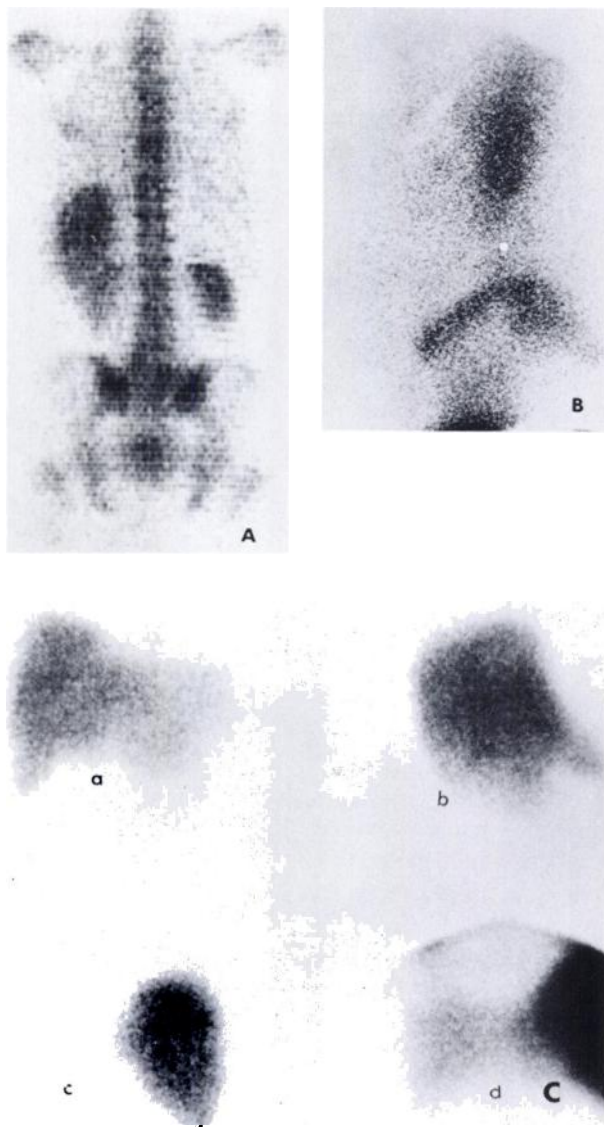
Splenic accumulation of bone-seeking agents has been reported in patients with Hodgkin's Disease and histiocytic lymphoma that involved the spleen (1,2). We would like to report a unique case of diphosphonate accumulation in a spleen containing metastatic adenocarcinoma and the simultaneous presence of functional asplenia.

A 57-year-old white female presented with pain of 3 mo

duration in the right hip and low back. Bone marrow biopsy revealed poorly differentiated adenocarcinoma cells. A search for a primary tumor was undertaken but extensive radionuclide and radiographic scans were all normal. A presumptive diagnosis of metastatic breast carcinoma was made and therapy was begun with fluoxymesterone and Prednisone. She responded with diminished bone pain and an increase in platelets from 22,000/mm<sup>3</sup> to 72,000/mm<sup>3</sup>, but she required intermittent blood transfusions for persistent anemia. One year following initial therapy she was readmitted with fatigue, anorexia, and abdominal pain. Reassessment included negative xerography and metastatic radiographic series. A Tc-99m diphosphonate bone scan showed no bone lesions, but there was a significant accumulation of radioactivity in the spleen (Figs. 1A and B). Although ultrasonic examination confirmed clinically evident splenomegaly, a Tc-99m sulphur colloid liver and spleen scan revealed absent splenic activity and no evidence of metastases to the liver (Figure 1C). A gallium scan did not show any accumulation within the spleen. Howell-Jolly bodies were found in the peripheral blood. Five months later bone scan and roentgenograms revealed metastatic disease to the bones. The concentration of diphosphonate and lack of colloid uptake in the spleen persisted. Autopsy revealed a diffuse infiltrating lobular carcinoma of both breasts, with metastases to the peritoneal cavity, both adrenal glands, hilar nodes, visceral pleura, lumbar vertebral bodies, and liver (multiple tumor nodules, 0.2-2 cm in diam of the right and left lobes). The spleen weighed 310 g and contained multiple 0.2-0.3 cm diam white nodules of pleomorphic cells. The red pulp was filled with golden-brown granular pigment that stained positive for iron.

The coincidental development of functional asplenia with nodular metastatic disease to the spleen was documented both by the peripheral smear containing Howell-Jolly bodies and absent activity on the Tc-99m sulphur colloid scan. Chaudhuri (1) reported splenic accumulation of radiostrontium in the spleen of a patient with Hodgkin's disease, and subsequently Winter (2) reported splenic accumulation of Tc-99m diphosphonate in a case of stage IV malignant histiocytic lymphoma. In neither of these cases was calcification or inflammation present in the spleen. In our case histologic and xerographic search for splenic calcification was also negative. Concentration of a bone scanning agent has been reported in three cases of metastatic colonic carcinoma to the liver (5). The tissue concentration of diphosphonate may be secondary to intracellular ion exchange with calcium phosphate. Usefulness of the phosphate scans in myocardial infarction relies on the exchange of the tracer for calcium ions in the damaged cardiac cells (6). Other factors, however, must play a role, since there were metastatic deposits within the liver that did not accumulate activity. In the patient we studied there was hemosiderosis of the liver and spleen, which has been shown to disturb the organ distribution of bone-seeking radiopharmaceuticals. Such patients usually show diminished skeletal uptake and increase in renal activity, however, without hepatic or splenic accumulation (7).

The association of sickle cell anemia and splenic accumulation of diphosphonate (with and without splenic calcification) has been well documented (3,4). On the other hand, the spleen may be enlarged with no uptake of the Tc-99m sulphur colloid. Our patient exhibited splenomegaly with an intact blood supply, but developed functional asplenia in the presence of metastatic tumor deposits. Explanations for the phenomenon in sickle cell anemia have included



**FIG. 1.** (A) Bone scan (posterior view) with Tc-99m diphosphonate, showing increased splenic accumulation. (B) Left lateral view of bone scan confirmed presence of activity within spleen. (C) Technetium-99m sulphur colloid liver and spleen scan showing no activity in spleen. Normal liver. (a) anterior, (b) right lateral, (c) posterior (centered toward right), and (d) posterior (midline).

(a) sluggish intrasplenic circulation secondary to the increased viscosity of the blood containing sickled cells, and (b) blockage of the reticuloendothelial system by oversaturation of these phagocytic cells (8). Possibly the severe hemosiderosis secondary to multiple transfusions bears some relation to the decreased splenic uptake of sulphur colloid.

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**A Modified FORTRAN Program for the Calculation of Modulation Transfer Function**

In a recent Concise Communication, Benedetto and Nusynowitz (1) presented a FORTRAN program for calculation of the modulation transfer function (MTF) from line spread function data. Several assumptions were made concerning the symmetry of the line spread function (LSF): specifically, that the peak value of the LSF occurs at the origin and that the LSF is symmetric about this peak value. Yet in their example the LSF is clearly not symmetric about the peak value. The true peak of the LSF curve does not occur at the observed maximum, and hence the origin should not be taken to be this value. Further, the program requires an odd number of LSF values. If the LSF data are symmetric, but the true peak is straddled by two equal data points, the program requires the loss of an important data point in order to provide an odd number of LSF values.

We have been using a modified program that is independent of symmetry about the origin and accepts an odd or even number of LSF values. Our program also computes the integral of the MTF,

$$\int_0^{\nu} MTF(\nu) \cdot d\nu,$$

from frequency 0 to the first spatial frequency where MTF( $\gamma$ ) reaches 0. This integral provides a convenient basis for the comparison of modulation transfer functions.

**Calculations.** Let N = number of LSF values, and let each be represented by f(X<sub>i</sub>), where X<sub>i</sub> is the displacement along the abscissa. If N is odd, let m = (N - 1)/2; if N is even, let m = N/2.

Then, for a given spatial frequency  $\nu$ , define

$$SC = \sum_{i=-m}^{N-(m+1)} f(X_i) \cos 2\pi\nu X_i,$$

$$SS = \sum_{i=-m}^{N-(m+1)} f(X_i) \sin 2\pi\nu X_i.$$

Let  $\beta = \tan^{-1} (-SC/SS)$ . If  $\beta < 0$ , let  $\beta = \beta + \pi$ . Then

$$MTF(\nu) = \left[ \frac{SC \cdot \sin(\beta) - SS \cdot \cos(\beta)}{\sum_{i=-m}^{N-(m+1)} f(X_i)} \right]$$