LIVER SCAN SHOWING INTENSE LUNG UPTAKE IN NEOPLASIA AND INFECTION

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Two cases of intense lung activity uptake during routine liver imaging are presented. One patient died 6 days after uptake was seen, and had Kupffer cell tumor, or liver angiosarcoma, at autopsy. The second patient with an acute infection superimposed on alcoholic hepatitis showed intense lung uptake on the tenth day of a sustained course of very high fever. A repeat liver scan after the patient became afebrile showed no lung uptake.

Recent reports (*1–4*) have related possible macrophage activity in the lung to the finding of intense lung uptake during liver scans. Two cases suggestive of this possibility are presented.

MATERIALS AND METHODS

The ^{99m}Tc-sulfur colloid used was prepared by the thiosulphate-perrhenate method (*5*). Searle Radiographics Pho/Gamma HP camera and Pho/Dot rectilinear scanner were used for imaging.

CASE REPORTS

Case 1. A 64-year-old man was hospitalized on April 9, 1974 because of epigastric pain. Laminectomy showed plasmacytoma of the spine in 1965 with atypical plasma cells on marrow aspiration. Placed on melphalan, splenectomy was done in 1965 when steroid-resistant pancytopenia developed. Spleen showed no multiple myeloma but atypical plasma cells in marrow were again found and maintenance melphalan was administered between 1965 and 1971. He remained well until the 1974 admission when, although vital signs and orientation were normal, he appeared drowsy with slurred speech and asterixis. Spider nevi, icterus, and enlarged liver were found. Hematocrit was 32% and WBC and differential were normal. Enzymes were SGOT 155, LDH 350, alkaline phosphatase 85 mU/ml, and serum leucine aminopeptidase was 90 I.U. Prothrombin time was 14.7 sec (10.3 sec control). Total protein was 6.2 gm%.

A ^{99m}Tc-sulfur colloid liver scan on the second hospital day showed no lung uptake and patchiness of liver uptake with a large posterior filling defect on right lateral scan (Fig. 1, upper left), which on a later scan did not concentrate ^{67}Ga-citrate. Liver biopsy showed only small areas of necrosis. Neomycin, prednisone, and magnesium citrate were started. On the ninth hospital day BUN previously normal rose to 60 mg%, and a ^{131}I-Hippuran renogram showed bilateral concentration compatible with either parenchymal stasis or obstruction. He became worse with increasing oliguria and on the 50th hospital day passed tarry black stools. A second ^{99m}Tc-sulfur colloid scan (Fig. 1) at this time showed intense lung uptake with increased activity in rib marrow.

At autopsy, angiosarcoma cells were seen in apposition to the surface of hepatic cells (Fig. 2A). The filling defect on scan was an area of massive necrosis. The tumor was concentrated in some areas forming masses but the needle biopsy had sampled a tumor-free area. Macrophages in clumps were seen in the lung sections (Fig. 2B) but there was no evidence of microemboli or other pathology in the lung. Angiosarcoma, a rare tumor, has been associated with possible vinyl chloride (*6*) carcinogenesis. This patient had no history of such exposure but a long exposure to melphalan, not associated with angiosarcoma to our knowledge.

Case 2. A 37-year-old man vomiting bright red blood and in impending delirium tremens was hospitalized on November 24, 1971. There had been two previous admissions in 1971 for alcoholic hepa-

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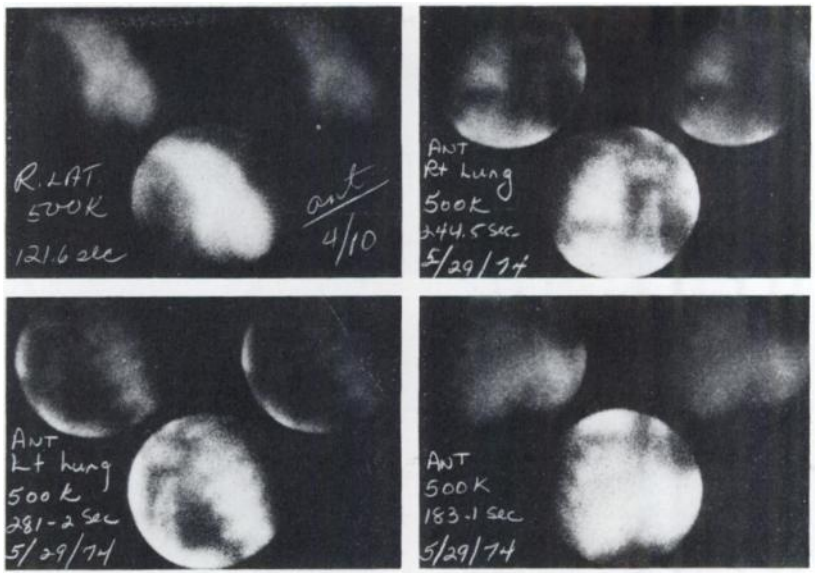

FIG. 1. Gamma camera image upper left shows large posterior filling defect as seen on first liver scan in Case 1. Other views show lung uptake of ^{99m}Tc-sulfur colloid activity, with high marrow activity.

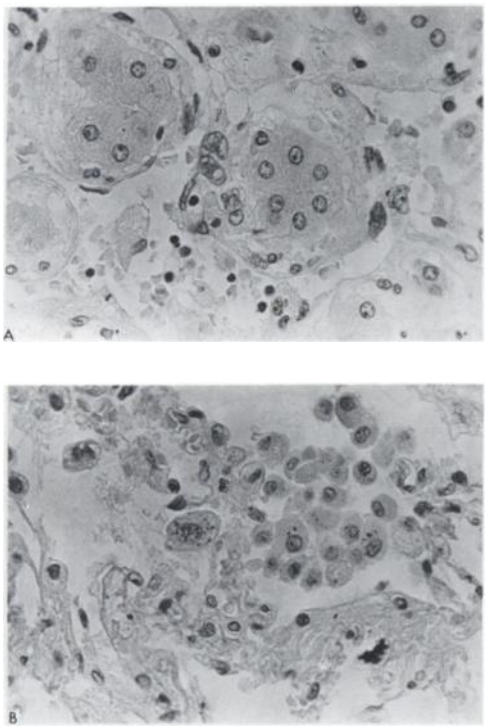

FIG. 2. (A) Angiosarcoma cells showing large, pleomorphic, bizarre-shaped nucleii surrounding hepatic cells. (B) Cluster of macrophages seen in lung adjacent to normal alveolar wall.

titis and gastritis. No spider nevi but ascites, enlarged liver, scleral icterus, and atrophic tongue papillae were present. Enzymes were SGOT 950, LDH 700, and alkaline phosphatase 150 mU/ml. Hematocrit was 42%, BP 150/96, and pulse 100/min. Intravenous fluids were started and, despite chloral hydrate and paraldehyde, he grew increasingly agitated and hallucinative but the bleeding stopped.

A left gluteal abscess developed at one of the injection sites. Temperature rose on the second hospital day to 104°F, and fluctuated between 102° and 105°F during the first 12 days. It was recorded on the chart at 105°F on the fourth, sixth, seventh, and ninth hospital days. From the fourth day the patient was in a cooling blanket and was given acetaminophen. The WBC was 8,500 mm^3 with 80% polymorphs, 14% lymphocytes, and 6% monocytes with normal platelets. On the seventh hospital day the abscess was drained and proved sterile, and since anaerobic infection was suspected, Loridine (2 gm per day) was started. Subsequently, ascitic fluid and other cultures done during the febrile period all showed no growth.

On the tenth hospital day a ^{99m}Tc-sulfur colloid liver scan showed intense lung uptake (Fig. 3). Highest recorded temperature on the day of the scan was 103.5°F and the lowest 102.0°F. A liver scan with radioactive colloidal gold was not done until the 15th hospital day when the patient's temperature was 98.6°F. It showed no lung uptake. The abscess at this time was granulating and much serosanguineous discharge had occurred during the previous 5 days. Either Loridine toxicity or hepatorenal syndrome with renal failure ensued with a BUN that peaked at 175 mg%. The patient improved gradually and was discharged well on the 64th hospital day with a BUN of 9.

DISCUSSION

Liver scans are routinely done using the same batch for four or five patients. There were three batch "controls" (*1*) that showed no lung uptake for the scans done with ^{99m}Tc-sulfur colloid. The radioactive gold colloid scan done on the second patient did not have batch "controls." One case in the literature (*2*)

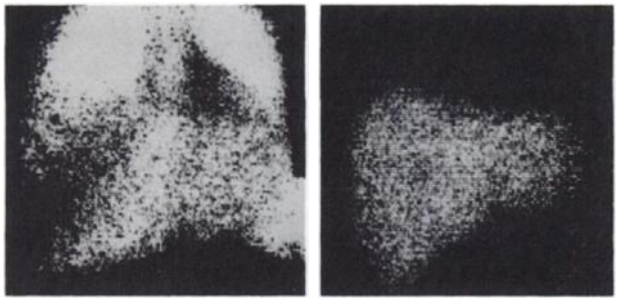

FIG. 3. Rectilinear scan image on left shows intense ^{99m}Tc-sulfur colloid activity taken up by lungs in Case 2. Separation of lung and liver is due to massive ascites. Five days later on right, radioactive colloidal gold liver scan showed no uptake in lung. Paracentesis had eliminated liver-lung separation.

showed lung uptake with both ^{99m}Tc-sulfur colloid and radioactive colloidal gold. If a colloid destabilizing factor (*1*) were present in the blood of these patients, capable of counteracting the lyophobic forces that keep colloidal particles apart, it might very well be capable of acting on a variety of different colloids. Possible conversion of the Tc_2S_7 colloid to a suspensoid, either macroaggregation of the colloid with lung trapping or its precipitation, or plating out in the lung vasculature might occur. One would then have to explain why the batch "controls" do not have the destabilizing factor. Colloids are known to aggregate on standing and gelatin is used as the stabilizing factor in the preparation of ^{99m}Tc-sulfur colloid.

Both of the cases reported here had elevated liver enzymes. Many of the cases so far reported showing lung uptake have had liver disease (*1,7,8*) with the type of uptake seen here. We have seen a case of extensive tubercular involvement of a portion of lung showing relatively low activity uptake of ^{99m}Tc-sulfur colloid activity with no observable activity elsewhere in the lungs, except in the area of the almost-coalesced lesions seen on x-ray. Several cases, one with amyloid (*8*), and another with lymphangitic lung metastasis (*9*) showed lung uptake, but the majority of cases so far reported showed no lung pathology.

It is possible that both lung macrophage and colloid destabilization may occur as the mechanism of lung uptake in different patients. Macrophages in large number were found in the lung sections in the autopsied case reported here but another reported case (*3*) with identical lung uptake did not show an inordinate increase in lung macrophages. Their presence does not prove they were responsible for the lung uptake.

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