

**LUNG UPTAKE OF <sup>99m</sup>Tc-SULFUR COLLOID DURING LIVER SCANNING**

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***Lung uptake of <sup>99m</sup>Tc-sulfur colloid was demonstrated in a patient with lymphosarcoma and hepatic cirrhosis as underlying diseases.***

Accumulation of <sup>99m</sup>Tc-sulfur colloid administered for liver scanning has previously been noted in patients who have received organ transplants or who have had extensive neoplastic infiltration of the lung or severe liver disease. We report a similar observation in a patient with lymphosarcoma and hepatic cirrhosis.

**CASE REPORT**

A 74-year-old man developed generalized lymph node enlargement 17 years previously and was seen by a surgeon who performed a biopsy of one of the inguinal lymph nodes. The histopathology showed lymphosarcoma and he received radiation therapy to the enlarged lymph nodes with complete regression of all lymph nodes. Subsequently the patient did very well until about 5 years ago when he was hospitalized due to extreme anemia that was found to be a Coomb's positive hemolytic anemia. He was treated with steroids and transfusions which controlled the hemolytic anemia. In 1968 he also showed elevation of the WBC to 28,000 with 83% lymphocytes. In December 1968 a rose bengal liver scan showed slow accumulation pattern suggestive of hepatitis, cirrhosis, or disseminated tumor and further studies were indicated to differentiate these possibilities. A bone marrow examination in October 1971 showed lymphoid infiltration of the bone marrow.

In July 1973, he was hospitalized with ascites and leg edema for further studies. A physical examination showed no peripheral lymphadenopathy. Liver and spleen were difficult to palpate. He had marked thrombocytopenia with a platelet count of 68,000. A bone marrow biopsy showed diffuse as well as nodular lymphatic infiltration.

In July 1973, he was referred for a liver scan which was performed with Squibb <sup>99m</sup>Tc-sulfur colloid (Tesuloid®) prepared according to instructions supplied in the kit.

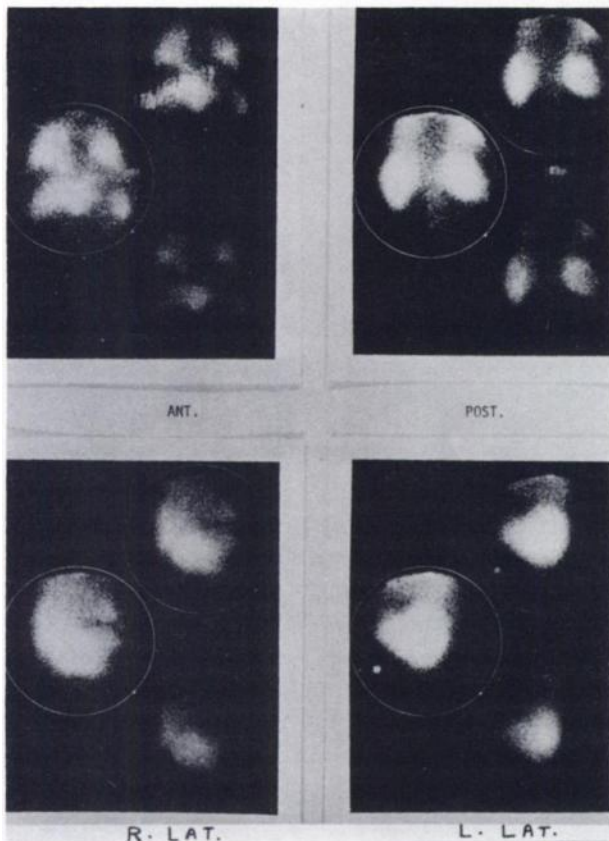
Over 500 liver scans have been performed in this department using the colloid prepared in the manner indicated. No previous example of accumulation of <sup>99m</sup>Tc-sulfur colloid in the lungs was detected.

The scans obtained 10 min after injection of 3 mCi of the colloid preparation are shown in Fig. 1. The liver-spleen scan shows splenic enlargement. The domes of the diaphragm are high and the liver is pushed upward in the abdomen. The irregularity of liver contour suggests possible abnormal infiltration. The lung uptake of the colloid is clearly visualized in the AP and PA views. In the left lateral view the enlarged spleen is apparent and in the right lateral view the liver and the lungs are seen. Enlarged spleen can also be seen in the PA view which imitates the positioning of a kidney scan.

**DISCUSSION**

Two patients before and one patient after this patient (SH) were also studied by liver scan. They were all injected with <sup>99m</sup>Tc-sulfur colloid from the same vial. The other three patients failed to exhibit any lung uptake of the <sup>99m</sup>Tc-sulfur colloid. Hence this rules out any drawbacks in the quality of the preparation or any formation of large particles in the vial due to aluminum ion contamination (1). A detailed study done on the effect of aluminum concentration in technetium eluant on particle size revealed no correlation between clinical results and aluminum content as high as 44.3 μg/ml in the eluant used in the preparation of the <sup>99m</sup>Tc-sulfur colloid (2). However, ionic aluminum (III) in generator

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**FIG. 1.** Lung uptake of  $^{99m}\text{Tc}$ -sulfur colloid. The images were obtained using gamma camera (Pho/Gamma II of Searle). Each view is photograph showing three separate images with varying degrees of contrast.

eluate as an erythrocyte-agglutinating agent is well known (3).

Before further studies could be done, the patient died 1 month after the liver scan was performed and autopsy showed lymphosarcoma with involvement of bone marrow, spleen, and the supraclavicular, mediastinal, para-aortic, iliac, and peripancreatic lymph nodes. It also showed severe macronodular cirrhosis of the liver, arteriosclerosis of the aorta and coronary arteries, ascites, and bilateral pleural effusion as well as small pericardial effusion. It did not reveal any multiple pulmonary microemboli.

Klingensmith, et al showed that lung uptake of  $^{99m}\text{Tc}$ -sulfur colloid is associated with transplantation of organs containing large numbers of intravascular macrophages (4) and that the lung uptake of  $^{99m}\text{Tc}$ -sulfur colloid was gradual with time, suggestive of a reticuloendothelial mechanism (5). This mechanism is not applicable to our present study. An unusually large number of macrophages present in the lung might phagocytize the colloid particles but autopsy findings negate this hypothesis.

Gillespie, et al (6) rejected the idea that lung uptake of  $^{99m}\text{Tc}$ -sulfur colloid may be due to severe

and long-standing liver disease (7). However, they felt cancerous infiltration in some way might have been responsible for the lung uptake but they could not do an autopsy due to denial of permission. In the present case, no such infiltration was found at autopsy.

Increased RES phagocytic activity was found in patients with both neoplasia and infection (8) and the mechanism of RES enhancement in neoplastic disease is not well understood.

The explanation of Keyes, et al (9) that the fact that the lungs represent the first site of RES activity encountered following intravenous injection of the colloid might be responsible for increased relative lung uptake is not satisfactory. They think there is insufficient evidence to suggest that lung uptake may be due to the rapid intravascular clumping of the injected colloid particles with subsequent entrapment in the pulmonary capillary bed. In the present case there was no evidence of multiple pulmonary microemboli at autopsy.

Kitani and Taplin proposed different labeling sites on the albumin molecule and catabolic processes in order to explain their observation of slow liver release rates of  $^{99m}\text{Tc}$ -albumin colloids compared with  $^{131}\text{I}$ -albumin colloids (10). They also clearly demonstrated the presence of a biliary pathway of  $^{99m}\text{Tc}$  excretion in man when  $^{99m}\text{Tc}$ -albumin colloid and microaggregates are administered intravenously but not for  $^{99m}\text{Tc}$ -sulfur colloid (11). This means that in a normal liver  $^{99m}\text{Tc}$ -sulfur colloid is phagocytized by the Kupffer cells and  $^{99m}\text{Tc}$  is excreted into the venous blood. We postulate that in certain patients due to pathophysiological reasons most or part of the  $^{99m}\text{Tc}$ -sulfur colloid injected might aggregate into macromolecules which would then be trapped in the lungs. Hence we are also of the opinion that this phenomenon might be an alteration in colloid physiology due to the presence of disease. Further investigations are warranted for a clear understanding of the mechanism.

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