Post-Traumatic Pulmonary Accumulation of Tc-99m Sulfur Colloid

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A case is reported in which accumulation of Tc-99m sulfur colloid in one lung occurred following trauma, although the lung remained clear radiographically. Another trauma case is mentioned in which the uptake was bilateral. The cause of this phenomenon is unclear; several potential mechanisms are discussed.

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The accumulation of Tc-99m sulfur colloid in the lungs during liver-spleen imaging has been previously cited in the literature (*1–6*). Although there is a long list of possible causes (Table 1), there has not been a previous report documenting the occurrence of this phenomenon following trauma to the lungs. We describe a case in which unilateral pulmonary sequestration of Tc-99m sulfur colloid was visualized following blunt injury to the chest.

CASE REPORT

During retrieval of a fly ball, a 21-yr-old baseball player incurred blunt injury to the left thorax and upper abdomen when he collided with a fence. Initially he complained of left lower chest and upper abdominal pain, but continued to play in the game for a short time until he collapsed. He arrived in the emergency room in shock, though the admission hematocrit was 43%. On radiographic examination of the chest there was no evidence of pulmonary parenchymal injury or fracture of regional bony structures (Fig. 1). The abdominal examination was normal. A 3-mCi Tc-99m sulfur colloid liver-spleen scan revealed multiple lacerations of the spleen associated with a very large hematoma displacing the spleen anteriorly. Diffuse accumulation of tracer was noted throughout the left lung field (Fig. 2). Surgery confirmed that the spleen was macerated and associated with a huge hematoma. On the third postoperative day a repeat Tc-99m sulfur colloid image showed a normal liver and no pulmonary uptake of radiocolloid (Fig. 3). A repeat chest radiograph remained normal. The patient was discharged following an uneventful postoperative course.

DISCUSSION

No other patients receiving doses from the same batch of Tc-99m SC showed lung uptake on the resulting scans. Therefore, factors other than technical (formulation) problems were responsible for pulmonary retention of the radiocolloid, particularly since it was unilateral. Since the lung fields in this patient remained clear radiographically, pulmonary contusion and/or hemorrhagic changes also seemed unlikely as a cause. We have observed lung uptake of Tc-99m SC following trauma on only one other occasion. This occurred in a patient who sustained multiple bilateral rib fractures and had a flail chest. Since sulfur colloid was visualized in both lungs, a series of liver-spleen studies was obtained in this patient. Disappearance of the pulmonary colloid accumulation was noted at 17 days following the initial study. During this 17-day period the lung fields remained clear radiographically without evidence of contusion, hemorrhage, or changes consistent with fat embolism or adult respiratory distress syndrome. Perhaps the mechanism of post-traumatic pulmonary radiotracer sequestration is similar in these two patients, differing only in the degree of trauma sustained.

Studies in animals and humans have suggested that deposition of fibrin and fibrin degradation products in the lungs could be a potential cause for pulmonary accumulation of Tc-99m SC (*1, 5*). Perhaps occult injury to the lungs in the case presented served as a stimulus for fibrin deposition and subsequent sequestration of radiocolloid. Alternatively, subclinical physical damage to the pulmonary capillary bed may have initiated an inflammatory response, resulting in adherence of radiocolloid particles to capillary endothelium (*7*) without concomitant trapping of blood cellular

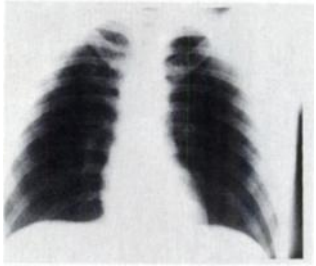

FIG. 1. Chest radiograph on day of injury. No abnormalities of lungs or skeletal structures were observed.

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TABLE 1. REPORTED FACTORS ASSOCIATED WITH PULMONARY UPTAKE OF TECHNETIUM-99m SULFUR COLLOID

Factors	
Al^{3+} (aluminum antacids)	Hepatocellular disease, hepatic failure, and/or intrahepatic cholestasis
Bacterial endotoxin	Liver angiosarcoma
Malignant lymphoma	Acute infection superimposed on alcoholic hepatitis
Spleen or bone marrow transplant	Children (? normal finding)
Intra-abdominal abscesses	Disseminated intravascular coagulation
Advanced breast carcinoma	Neoplasia (various)
Mucopolysaccharidoses Type II (Hunter)	Systemic amyloidosis
Falciparum malaria	Intravascular clot
Histiocytosis X	Exogenous RES stimulants
Liver transplant	Atelectasis (focal uptake)
Variation in colloid preparations (e.g., macroaggregation of the radiopharmaceutical and other technical factors)	

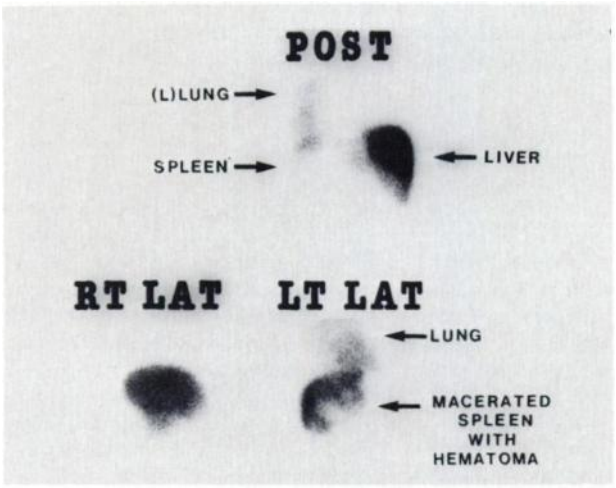

FIG. 2. Tc-99m sulfur colloid liver-spleen scan. Posterior and both lateral images on day of injury show uptake of radiocolloid in left lung.

components. It is known that both inflammation and stress give rise to an increased number of circulating macrophages (*8–10*). Possibly these cells circulate to pulmonary capillaries and surrounding interstitial tissue where they retain the ability to phagocytize colloidal material.

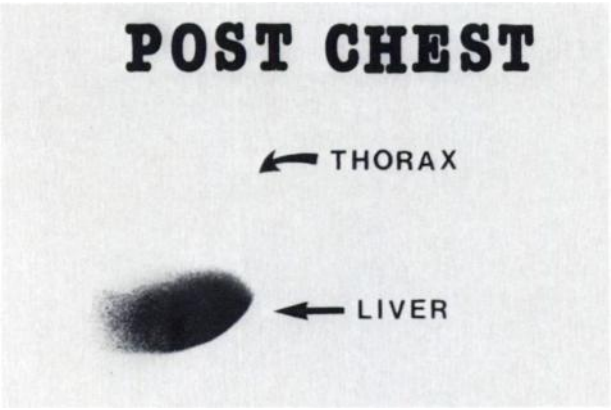

FIG. 3. Posterior image of repeat Tc-99m SC study three days after trauma and splenectomy. There is no localization of radiotracer in lungs and splenic activity is absent.

Another hypothesis is that ipsilateral hypoventilation due to splinting resulted in slowing of pulmonary circulation with a corresponding delay in the clearance of Tc-99m SC from the left lung.

In conclusion, regardless of the specific underlying mechanism, trauma may be recognized as an additional cause for lung uptake of Tc-99m sulfur colloid, though its occurrence is considered rare.

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