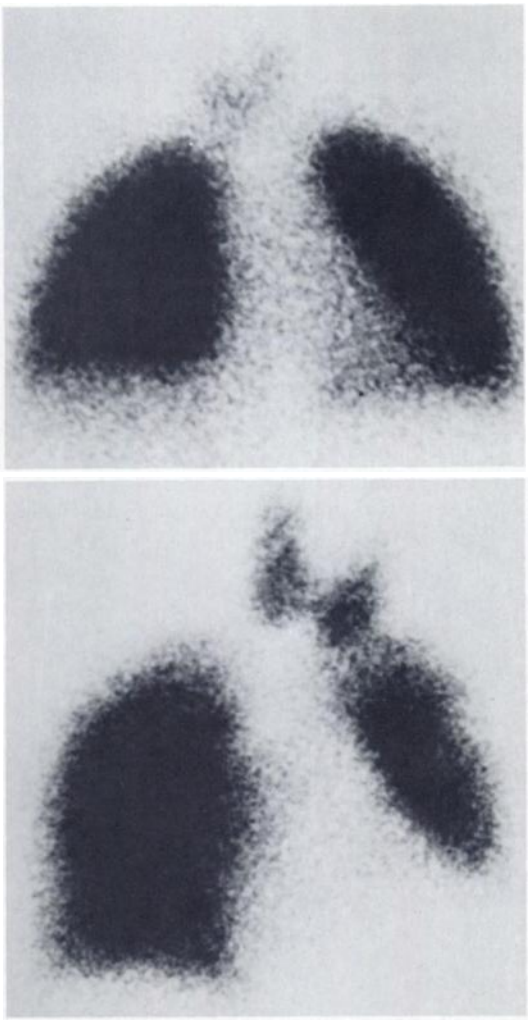

FIG. 1. Perfusion lung scan performed 20 min after i.v. injection of 4 mCi of Tc-99m macroaggregated albumin demonstrates normal activity in lungs, and also activity in thyroid because of thyroiditis (top) and Graves' disease (bottom).

otherwise showed no abnormalities except for diffuse thyromegaly without nodularity. A neurological examination showed rapid relaxation time of the deep tendon reflexes and slight proximal muscle weakness.

Clinical diagnosis was that of thyrotoxicosis. Thyroid studies showed a TSH of 3.6 μIU/ml, resin T_3 uptake of 55%, thyroxine of 18 μg/dl, and total T_3 by radioimmunoassay of 412 ng/dl.

During the hospital course, respiratory distress with pleuritic chest pain led to a lung image to search for pulmonary embolism. The perfusion image, performed after the intravenous administration of 4 mCi of Tc-99m macroaggregated albumin, excluded pulmonary embolism but demonstrated a large amount of thyroid activity (Fig. 1, bottom). There was no uptake in the brain or kidneys to suggest a right-to-left shunt, nor activity in salivary glands or stomach to suggest large amounts of free pertechnetate. Thyroid uptake and scan with 200 μCi of iodine-123 demonstrated 100% uptake at 24 hr and an enlarged gland with diffuse uptake consistent with Graves' disease.

These two cases demonstrate thyroid activity on lung imaging with Tc-99m macroaggregated albumin, due to thyroid disease. Macroaggregated albumin injected intravenously is nearly completely trapped in the precapillary arterioles of the lung, with little activity seen in other organs (*1*,*2*). When there is a right-to-left shunt, either intracardiac or intrapulmonary, the particles gain access to the systemic circulation and may be seen in brain and kidneys (*3*). Our patients had no right-to-left shunt, nor activity in brain or kidneys.

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The other situation in which activity can be seen in organs other than lung is when there are high levels of free pertechnetate due to poor quality control. In this situation one would find activity in salivary glands and gastrointestinal tract, but this was not seen in our patients. Furthermore, other perfusion lung images performed on different patients during the same period showed no thyroid activity, and the radiochemical purity of our radiopharmaceutical was greater than 99%.

The thyroid gland is well known to accumulate free pertechnetate in both normal and diseased states (*4*,*5*). Despite the small amount of free Tc-99m found in our compound, calculated to be less than 0.05 mCi (<1%), there was intense thyroid uptake due to TSH stimulation secondary to the hypothyroidism resulting from Hashimoto's thyroiditis in the first patient, and to the thyroid-stimulating antibodies of Graves' disease in the second.

These cases show that thyroid activity due to thyroid disease may be seen during perfusion lung images, and should be included in the differential diagnosis of extrapulmonary uptake. Recognition of this could lead to the diagnosis of unsuspected thyroid disease.

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Lung Uptake of Tc-99m-Tin Colloid in a Patient with Lassa Fever

Significant pulmonary uptake of Tc-99m-tagged tin- or sulfur colloid is normally less than 1% of the injected dose, whereas the liver extracts 80%, the spleen 15%, and the bone marrow 5% (*1*). It is now established, however, that several diseases are associated with increased colloid in the lungs, and that the prevalence of this phenomenon is approximately 1.6% among routine liver scans (*2*). We wish to report the liver-scan appearances of a patient with Lassa fever (a rare arenovirus infection) in whom significant lung uptake of Tc-99m tin colloid was also observed. The clinical and virological features of this patient's disease (*3*), and the ensuing community surveillance problems (*4*), have been reported in detail elsewhere.

An 18-yr-old Nigerian girl was admitted to our hospital in January 1982, having arrived in London from her native country

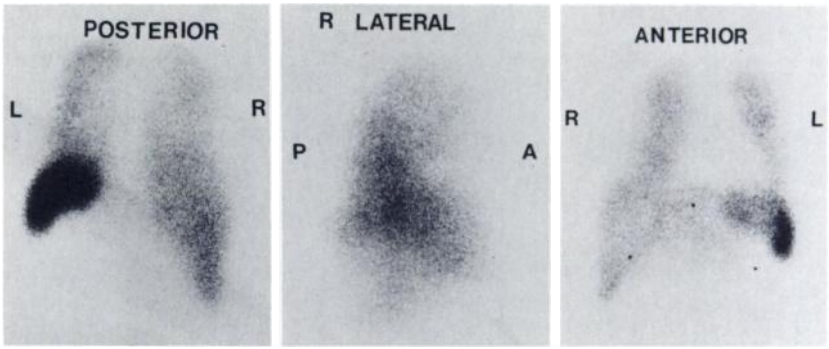

FIG. 1. Tc-99m tin colloid liver/spleen scan. Posterior, right lateral, and anterior views, showing abnormal uptake of colloid in lungs. Costal margins are marked by dots on anterior view.

twelve days previously. For eight days before admission, she had experienced night sweats, frequency of micturition, and intermittent abdominal colic relieved by bilious vomiting. Physical examination revealed a febrile (38°C) lady who appeared relatively well apart from mild epigastric tenderness. No purpura, pharyngeal exudate, hepatosplenomegaly, nor impairment of higher mental functions were noted. Initial investigations were unremarkable, including repeated examination of blood films for malarial parasites. Liver function tests were normal except for the alanine transaminase, which was 266 units/l (normal < 20). Her clinical state was essentially unchanged until the fourth hospital day, when she began to vomit frequently. The alanine transaminase rose to 850 units/l, the alkaline phosphatase to 45 KAU/dl (normal < 11), and the aspartate transaminase to 4,440 units/l (normal < 18). Hepatitis B antigen screen and hepatitis A specific IgM were negative. Upper abdominal ultrasound suggested an echogenic area to the left of the midline. A Tc-99m tin colloid liver image demonstrated reduced, patchy uptake in an enlarged liver, with increased uptake in an enlarged spleen (Fig. 1). There was also significant lung uptake of tracer, not due to technical difficulties in the preparation of the radiopharmaceutical. By the fifth hospital day, her general condition was deteriorating, and no diagnosis had been reached despite extensive investigation. A diagnosis of Lassa fever was still considered possible, and in view of the risk of uncontrollable hemorrhage (and thus spread of infection), she was transferred to a high-security unit for infectious diseases. Subsequent investigations confirmed the diagnosis of Lassa fever, and she was treated with Lassa convalescent serum. She remained seriously ill for 1 mo but was eventually discharged well on the 60th hospital day.

Our patient's illness is an example of a very rare, severe viral infection, associated with liver inflammation and giving rise to marked lung uptake of tracer colloid. Three mechanisms have been put forward to account for the increased lung uptake of colloid: increased reticuloendothelial system activity, intravascular clumping and embolization, and excessive Al^{3+} ions resulting in flocculation and thus pulmonary embolism (*5*). Based on the first of these, a plausible explanation of lung uptake of colloid in patients with liver disease is that endotoxin, which is normally removed from portal blood by Kupffer cells in the liver, escapes into the systemic circulation. It stimulates the bone marrow to release phagocytic cells, which then lodge temporarily in the pulmonary capillaries (*6,7*). Against this theory is the observation that lung uptake is seen in patients with cirrhosis who do not have bone-marrow uptake (*2*), since the reticuloendothelial cells of the marrow would normally be expected to be as avid for colloid as the lung macrophages. However, all of the injected colloid must pass through the lungs before reaching the bone marrow; thus all the colloid is available for extraction by the lung macrophages before the bone-marrow phagocytes are exposed to it. Moreover, on each subsequent recirculation of untrapped colloid that has not been phagocytosed during the first pass, all of it will pass through the pulmonary circulation, whereas only a small fraction will pass through the bone-marrow capillaries.

In conclusion, we report details of a liver image showing lung uptake of colloid, in a patient who survived an episode of Lassa fever, a rare arenovirus infection with a significant mortality (*8*). The exact mechanism responsible for the unusual scan appearances is still disputed, but endotoxin escape into the systemic circulation with accumulation in the lungs of phagocytes originating in the bone marrow, is a plausible explanation on the available evidence.

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