

RENAL UPTAKE OF ^{99m}Tc -SULFUR COLLOID

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Accumulation of ^{99m}Tc sulfur colloid by the kidneys was observed in two patients with congestive heart failure. In one instance renal uptake of ^{67}Ga -citrate was also observed.

Technetium-99m-sulfur colloid, after intravenous administration, is extracted from the plasma primarily by the reticuloendothelial system (RES) of the liver and to a lesser extent by the RES of the spleen, bone marrow, and other organ systems (1-4). Using radiocolloids, the liver and spleen are clearly visualized. In contrast, no uptake is normally seen in the kidney and lung (1-4). Indeed, by monitoring the urine for 24 hr after the intravenous administration of ^{99m}Tc -sulfur colloid, Patton, et al have demonstrated that less than 0.2% of the nuclide is excreted through the kidneys (2). Recently we have observed two patients in whom distinct visualization of the kidneys occurred during liver scanning. They are the subject of this report.

CASE REPORTS

Case 1. SV, a 55-year-old white man, was admitted with an 8-year history of progressive dyspnea on exertion, paroxysmal nocturnal dyspnea, orthopnea, ascites, and edema. There was no history of previous myocardial infarction, rheumatic fever, familial heart disease, alcoholism, hepatic or renal disease.

On physical examination, the patient displayed mild respiratory distress with a blood pressure of 118/102, pulse rate of 100/min, and respiratory rate of 30/min. There was marked anasarca. Jugular venous pulse was distended to the angle of the jaw with the patient 90 deg upright. The heart was palpably enlarged with audible S3 and S4 gallop rhythms. Murmurs characteristic of mitral insufficiency and tricuspid insufficiency were present. The liver was enlarged and pulsatile.

Laboratory studies demonstrated a hematocrit of 45%, white blood cell count of 5,900, and a sedimentation rate of 2. The BUN, serum creatinine, and urine analysis were normal. Liver function tests showed an SGOT of 51 units, SGPT 28 I.U., alkaline phosphatase 5.4 Bodansky units/ml, and total serum protein 6.8 gm% with albumin of 3.5 gm%. The patient underwent cardiac catheterization and the end-diastolic pressure was increased in both the right and left ventricle and the cardiac index was profoundly reduced to 1.57 liters/min/meters². The peripheral vascular resistance was markedly elevated to 378 dynes/sec/cm⁵ and the A-VO₂ difference increased to 6.3 vol %. The coronary arteries were normal and there was mitral and tricuspid regurgitation.

During this admission, three separate ^{99m}Tc -sulfur colloid liver-spleen scans were performed after the intravenous injection of 5 mCi of the radiopharmaceutical (Fig. 1). Each demonstrated slightly mottled uptake of the radiopharmaceutical in a normal size liver, splenomegaly, slight uptake by the bone marrow, and intense homogeneous uptake by the kidneys. There was evidence of ascites with the liver and spleen being slightly displaced from the body wall by fluid.

In order to help exclude unsuspected anomalous vascular pathways to the kidneys, a radionuclide angiogram using ^{99m}Tc -sulfur colloid as the radiopharmaceutical was performed by injecting a dorsal foot vein. No evidence of a vascular anomaly was noted. Further renal workup included a radionuclide dynamic study and a renal scan using ^{99m}Tc -penicillamine. A radionuclide renogram was also performed. These studies were entirely normal. A ^{67}Ga scan of the kidneys was also normal.

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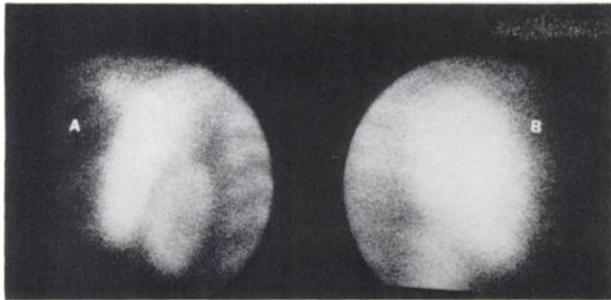


FIG. 1. Posterior scintiphotograph of spleen (A) and liver (B) demonstrating splenomegaly, slightly mottled uptake in normal size liver, activity in bone marrow, and homogeneous uptake in kidneys bilaterally. Note that renal intensity is similar to that of bone marrow.

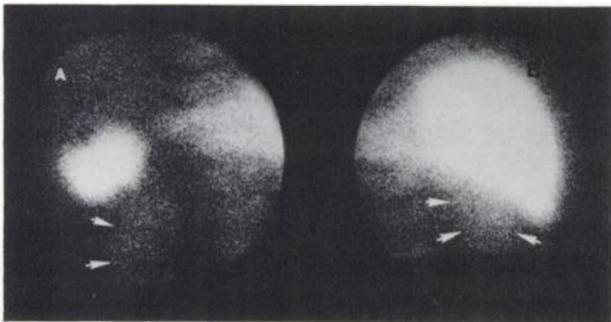


FIG. 2. Posterior scintiphotograph spleen (A) and liver (B) revealing homogeneous uptake in normal size liver, prominent splenic uptake, and minimal uptake in kidneys (arrows) and bone marrow.

Case 2. ID, a 62-year-old white man, was admitted with increasing shortness of breath, orthopnea, and marked peripheral pitting edema. He had a 15-year history of angina and several myocardial infarctions eventuating in severe chronic congestive heart failure. In the months preceding the current admission, the patient experienced increasing dyspnea and weight gain in spite of adequate digitalization and large doses of diuretics. In addition, he had a history of nephrolithiasis for which extensive evaluation failed to provide an etiology. There was no history of alcoholism or liver disease.

On physical examination, blood pressure was 120/80 and pulse rate was 80/min. There were bilateral basilar rales, a 13-cm jugular venous pulse in the erect position, and a palpably enlarged heart. S3 and S4 gallop rhythms and a grade I/VI systolic ejection murmur were present on auscultation. The liver was palpably enlarged, peripheral pitting edema was present, but no ascites was detected.

The laboratory results revealed a normal CBC. The BUN was 30 mg% and the serum creatinine was 1.5 mg%. The urinalysis was within normal limits. Liver function tests showed an SGOT of 22 units, alkaline phosphatase 4.5 Bodansky units, total bilirubin 0.8 mg%, total serum proteins 7.1 gm%

with an albumin of 3.1 gm%. Chest x-ray showed massive cardiomegaly with four-chamber enlargement and congestive heart failure. An IVP revealed a left ureteral calculus and moderate left hydronephrosis. Cardiac catheterization was not performed. A liver-spleen scan after the intravenous injection of 5 mCi of ^{99m}Tc -sulfur colloid demonstrated homogeneous hepatic uptake in a normal size liver, increased splenic uptake, minimal bone marrow uptake, and uptake in both kidneys (Fig. 2). A ^{67}Ga scan of the kidneys performed several months earlier revealed abnormal gallium uptake of both kidneys.

DISCUSSION

The ^{99m}Tc -sulfur colloid used in the present study was prepared by a modification of the method of Patton, et al (2). Since some renal or lung uptake may conceivably occur during ^{99m}Tc -sulfur colloid scans because of faulty preparation, we considered the possibility that such might be the case in the two studies presented. Accordingly, multiple studies were performed in one patient (Case 1) resulting in identical findings, thereby eliminating faulty preparation as the explanation. As further evidence, several other liver scans were performed using the same colloid preparation on the same day as the studies currently reported and in no other instance was even faint visualization of the kidneys detected.

Minimal renal uptake and excretion of ^{99m}Tc -sulfur colloid following intravenous administration of this radiopharmaceutical has been reported by others (1,2,5-7). In these instances, however, renal uptake has been in the range of 1% of the injected dose and has not been sufficient to allow imaging.

Several recent reports have called attention to visualization of the lungs with ^{99m}Tc -sulfur colloid in some patients after liver transplantation (8-10), in one additional patient after a spleen and bone marrow transplant (10), and in a patient with a malignant lymphoma (10). In these reports there is no mention nor do the scans reveal renal uptake of the radiopharmaceutical (8-10). It has been proposed that the uptake of the labeled colloid by the lungs in certain situations is due to an increase in the number of macrophages in the lungs as a result of migration of reticuloendothelial cells to the lungs after being released from the transplanted liver or spleen (9,10).

In standard radiocolloid liver-spleen scans of normal patients, it is rare to visualize other organs. In the event that the liver is unable to clear the colloid from the plasma at its usual rate, phagocytosis of the particles can occur in other RE sites (1-3). The kidneys, however, contain only a small amount of

interstitial connective tissue with a few fibroblasts and fixed macrophages. Furthermore, massive migration of reticuloendothelial cells to the kidneys is not known to occur as a response to common disease states (10,11).

It is of interest that both of these patients had profound right- as well as left-sided congestive heart failure. The significance of this finding in a cause-effect relationship in the renal uptake of the colloid is difficult to evaluate since, in our experience, it has not been observed in other patients with severe right and/or left heart failure.

Although there was evidence of mild hepatocellular disease in both patients, the amount of uptake of the colloid by the kidney was far more intense than the bone marrow uptake in either case and therefore decreased hepatic uptake would not appear to be a tenable explanation for the renal accumulation of the colloid. Our observations suggest the possibility that an as yet unidentified mechanism may exist in some cases of severe congestive heart failure which promotes uptake of ^{99m}Tc -sulfur colloid by the kidneys. The authors would appreciate knowing of the experience of other investigators with regard to the renal uptake of radiocolloids.

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