jnm / CASE REPORT

RENAL COLLOID LOCALIZATION

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Renal uptake of ^{99m}Tc-sulfur colloid was observed in a patient with cardiac failure, pleural effusion, and ascites. Colloid labeled with ^{113m}In gave similar results.

The organ distribution of intravenously administered ^{99m}Tc-sulfur colloid has been well documented. Under normal circumstances, in several experimental animals, about 75–90% of the colloid localizes in the hepatic Kupffer cells, 1–3% in the spleen, and 3–8% in the bone marrow (*1–3*). The relative distribution within these tissues is altered in a variety of pathogenic states including diffuse hepatic disease and hematologic disorders (*4*). Recently, several authors have reported increased pulmonary localization of colloidal radiopharmaceuticals not attributable to technical factors with resultant particle macroaggregation (*5–7*). We have recently encountered a patient with renal localization of both ^{99m}Tc-sulfur colloid and ^{113m}In-colloid; this phenomenon has not been previously reported to our knowledge.

CASE REPORT

A 52-year-old man was admitted to Barnes Hospital in July 1972, with fever, purpura, and gangrene of his left foot. Examination revealed a temperature of 40.4°C, blood pressure of 92/70, and pulse of 142. The remainder of the examination was unremarkable except for multiple purpura, a liver extending 4 cm below the right costal margin, and gangrenous changes in his left foot. Hematologic studies on admission revealed a white blood count of 14,500, hemoglobin of 10.2 gm%, hematocrit of 30%, and a platelet count of 30,000. Liver function studies revealed abnormal total bilirubin (1.8 mg/100 ml), SGOT 271 units, LDH 371 units with a normal alkaline phosphatase. Renal studies included a urinalysis on admission which revealed 1+ protein, a normal BUN and serum creatinine. During his complicated hospital course, he was shown to have streptococcal (Group A, beta hemolytic) septicemia, purpura fulminans, and a septic embolus causing gangrene of his left foot. He received intravenous penicillin; the fever and purpura resolved. His left leg was amputated below the knee. During this hospitalization a liver-spleen scan with ^{99m}Tc-sulfur colloid was performed to evalute for a possible hepatic abscess and was normal (Fig. 1). Laboratory data before discharge revealed all hematologic and liver and renal studies to be normal.

He was well until May 1973 when he was readmitted in congestive heart failure with pleural effusion and ascites. On admission his liver function tests were again abnormal with a total bilirubin of 2.0 mg/100 ml, SGOT of 54 units, LDH of 305 units, and total alkaline phosphatase of 159 units. His complete blood count, BUN, serum creatinine, urinalysis, and IVP were normal. With digitalis and diuretic therapy, his cardiac failure improved. Repeat liver function tests were normal. A repeat liver-spleen scan was requested to evaluate hepatomegaly and showed renal uptake of colloid (Fig. 1).

DISCUSSION

The pathogenesis of lung uptake of colloid has not been established. Experimentally, significant lung uptake of ^{99m}Tc-sulfur colloid has been demonstrated in mice after reticuloendothelial stimulation accompanying intraperitoneal endotoxin administration (*8*). Recently, lung uptake has been reported to be a nonspecific accompaniment of liver disease in patients with a variety of underlying diseases (*6*). Whether the lung uptake represents increased reticuloendothelial activity in the lung or a local change in the colloid has not been determined (*6*).

In animal distribution studies, less than 1.5% of

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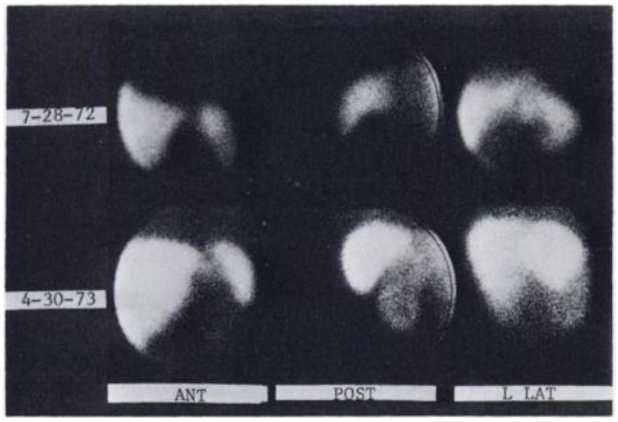

FIG. 1. Images of spleen showing abnormal area of localization inferomedial to spleen on 4–30–73 which was not present on 7–28–72.

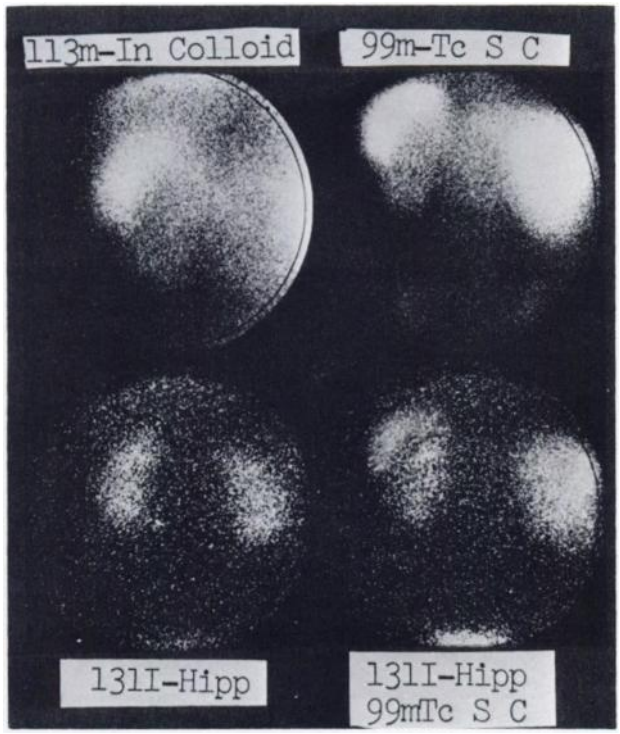

FIG. 2. Posterior images of spleen with abnormal area of ^{113m}In-colloid and ^{99m}Tc-sulfur colloid localization inferior to spleen. Posterior kidney images (^{131}I-Hippuran) and combined kidney-spleen image (^{131}I-Hippuran, ^{99m}Tc-sulfur colloid) show abnormal colloid localization coincides with kidney.

an injected dose of ^{99m}Tc-sulfur colloid normally localizes in the kidneys (*1,3*). Normally, there are no macrophages in the kidney (*9*) and there has been no evidence to suggest Kupffer cell migration from liver to kidney as has been shown to occur from liver to lung (*10*).

The ^{99m}Tc-sulfur colloid study at the time of this patient's first admission was normal (Fig. 1). When repeated 10 months later, an accumulation of activity was noted inferomedial to the spleen. An ^{113m}In-colloid study (Fig. 2) confirmed this finding as did a repeat examination with ^{99m}Tc-sulfur colloid (Fig. 2). In addition, ^{131}I-Hippuran renal images showed that the region of abnormal colloid localization coincided with the left kidney. The right kidney may also have had colloid localization but was obscured by the liver.

The mechanism of renal colloid localization in this patient remains enigmatic. Other patients studied on the same days with the same colloid preparations showed no renal activity. Images of the neck obtained after ^{99m}Tc-sulfur colloid administration in this patient revealed no thyroid or salivary localization, essentially excluding a substantial quantity of free $^{99m}TcO_4^-$ ion in the radiopharmaceutical preparation. The similar results with ^{113m}In-colloid further support our contention that radiocolloid was localized to the kidneys. Whether the colloid localization by the kidney represents reticuloendothelial function within the kidney or results from some in vivo alteration of the colloid is not known.

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